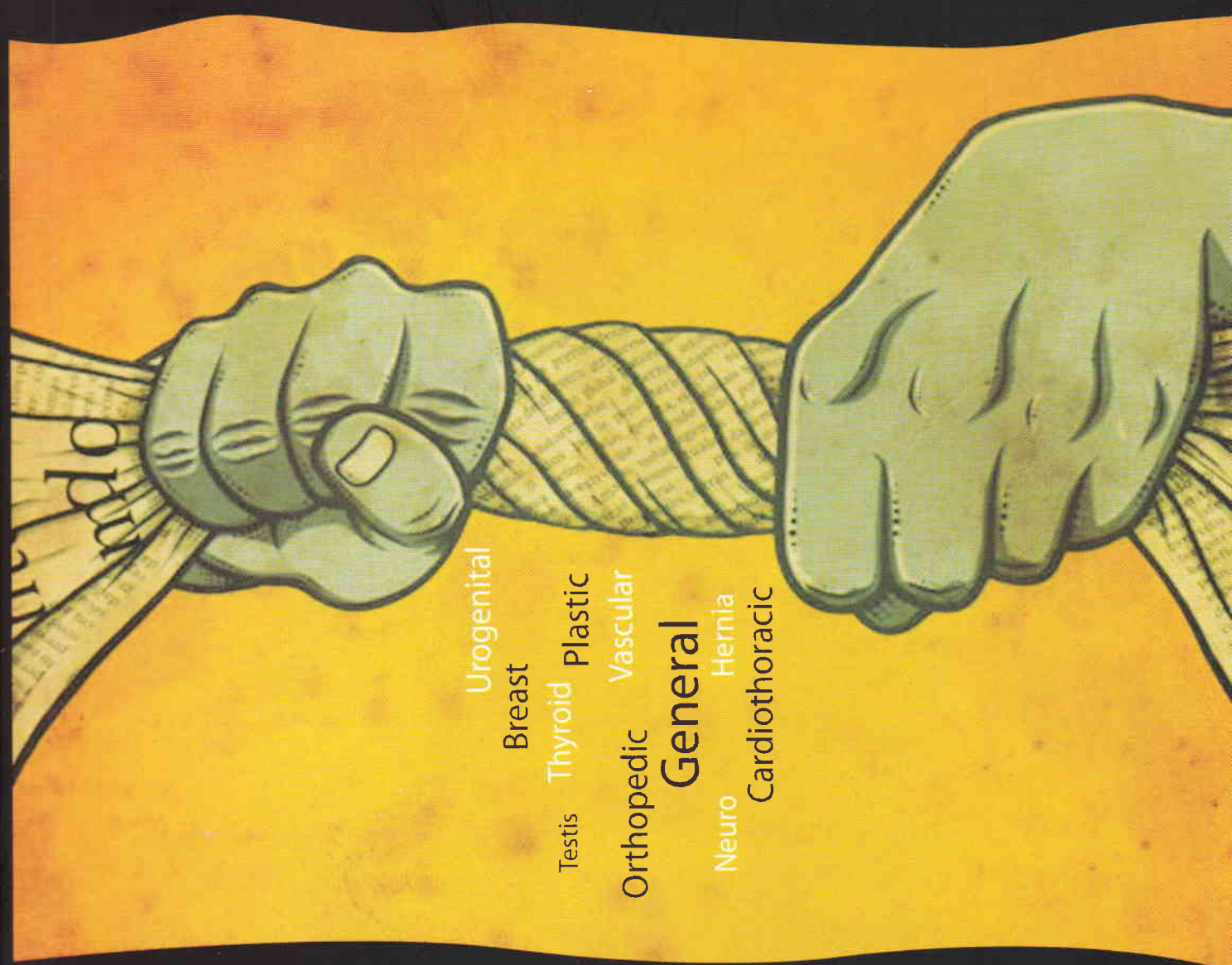


Matary Book of

SUMMARY

Mohammed El-Matary



Urogenital

Breast

Thyroid

Plastic

Testis

Vascular

Orthopedic

General

Hernia

Neuro

Cardiothoracic

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Preface

This book provides an update for medical students who need to keep abreast of recent developments. I hope also it will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you, the reader, will enjoy going through these pages as much as he had.

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1

GENERAL SURGERY



Ulcers of the Tongue

Classification

- A. Traumatic: frenal & dental ulcers
- B. Inflammatory:
 - i. Acute: dyspeptic, herpetic & lichen planus (autoimmune) ulcers.
 - ii. chronic: T.B. & syphilitic ulcers
- C. Malignant ulcer.
- D. Leukoplakia ulcer.

	Dental	Dyspeptic (Aphthous)	TB	Syphilitic
Etiology	Opposite sharp or broken tooth	Unknown	open pulmonary TB	3rd stage syphilis (Gumma)
Site	side of the tongue	superficial ulcer in all tongue & mouth	tip of tongue	Dorsum of tongue
Shape	Oval	Rounded or oval	Oval	Circular or serpiginous
Number	Single	Usually multiple	usually multiple	Single
Signs	painful	- Very painful - Associated with dyspepsia	very painful	Painless
Edge	Sloping	Thin punched out	Undermined	Punched out (EAO)
Margin	Hyperemic	Hyperemic	Cyanotic	Soft
Floor	Unhealthy granulation	Yellowish	Pale yellow granulation tissue	Leathery slough
Base	Soft	Soft	Soft	
LN's	With 2ndary infection	With 2ndary infection	Enlarged, soft & matted	With 2ndary infection
Management	- Remove the cause - Biopsy when malignancy is suspected	heals within 1-2 wks needs no specific treatment except for mouth wash & treatment of dyspepsia	- Sanatorial treatment - Anti TB drugs	Penicillin

	Frenal (post pertussis)	Herpetic	Leucoplakia	Malignant
Etiology	children with whooping cough → protruded tongue during cough	Unilateral eruption of small vesicles on the tongue → rupture → ulcer	Desquamation of the keratinized layer Due to 4S	1. Leucoplakia. 2. Papilloma. 3. <u>S</u> epitic teeth. 4. Smoking. 5. <u>S</u> pices. 6. <u>S</u> pirits.
Site	undersurface of the tongue	don't cross the mid line (unilateral)	Superficial ulcers	Sides of tongue
Shape	transverse linear ulcer	Multiple small rounded ulcers	Linear ulcers	Oval, rounded, irregular
Sum	Single	Multiple	Multiple	Usually single
Signs	Painful	Very painful	Painful	Painful+ Ankyloglossia Foeter oris
Edge	Sloping	Sloping, irregular	Fissure	Raised everted
Margin	Congested	Congested	Congested	Indurated
Floor	Granulation tissue	Granulation tissue	Granulation tissue	Necrotic
Base	Not indurated	Not indurated	Not indurated	Indurated
LN	Enlarged, elastic	Enlarged, elastic	Enlarged, elastic	Enlarged, hard&fixed
Manage-ment	TTT of whooping cough	Gentian violet paint	Avoid PF → 4S Biopsy	See cancer tongue

Carcinoma of Tongue

Incidence

- Anterior $\frac{2}{3}$: 50%
- posterior $\frac{1}{3}$: 20%
- Male > female .
- Age : >60years

Etiology

► Predisposing factors:

1. Leucoplakia.
2. Papilloma.
3. Sepitic irregular teeth.
4. Smoking.
5. Spices.
6. Spirits.
2. Syphilis in its tertiary stage.

Pathology

- **Site** → lateral side of tongue
- **Macroscopic & Microscopic** → as SCC (95%), Adenocarcinoma (5%)
- **Spread**
 1. **Direct** → tongue → floor of the mouth → mandible.
Tumors of the posterior $\frac{1}{3}$ spread to the tonsils, pharyngeal wall and larynx. They may cross to the other side of the tongue

2. Lymphatic:

- Tip → submental L.Ns → bilaterally to the submandibular and upper deep cervical LNs..
- Margin → submandibular L.Ns on same side. Those near the midline disseminate bilaterally
- Posterior 1/3 spreads directly to the upper deep cervical LNs.

3. Blood → Late to lung & bones.**Clinical Picture**

The classic clinical picture of tongue cancer is that an old man sitting in the outpatient clinic with cotton wool in his ear, and blood stained saliva dribbling from the mouth

Early cases

- The disease is symptomless. The patient may complain of persistent ulcer

Late cases

- Pain in tongue, 1st due to infection, later due to lingual nerve affection. Pain is referred to the ear via the auriculo-temporal nerve. In tumors of the posterior third pain may be felt on swallowing.
- Salivation. It may be blood stained and smells badly.
- Difficult speaking.
- Enlarged cervical LNs.
- Ankyloglossia.

Complications

1. Inhalation pneumonia.
2. Secondary hemorrhage.
3. Cachexia due to starvation.
4. Asphyxia.

Investigations

- **Routine.....**
 - **Diagnosis** → Biopsy → best is excisional with one cm safety margin.
 - **FNAC** of suspected cervical LNs
- **Staging.....**

Treatment

- Surgery and radiotherapy are the main lines of treatment.
- Chemotherapy is used as an adjuvant in some cases.
- Radical treatment of early cases

A. SURGERY:

- Resection procedures
 - Carcinoma in situ needs excision with 1cm safety margin.
 - Carcinoma of the anterior two thirds of the tongue is excised with 1.5 cm safety margin..
 - Carcinoma of the posterior third is treated either by total glossectomy.
 - If there are lymph nodes metastases, they are excised by complete neck dissection..
 - If the mandible is affected , it must be excised known as Comandó operation.

B. RADIOTHERAPY:**Indications**

- T1 and T2 (less than 4cm) may equally benefit from surgery or radiotherapy.
- Palliation for advanced cases

Methods

1. Radiotherapy.
2. Palliative resection of the primary if possible may comfort the patient.
3. Analgesics, nasogastric feeding or tracheostomy may be required.
4. Chemotherapy.

Prognosis

- 1- TNM staging. Lymph node involvement is the most important prognostic index.
- 2- Prognosis is better in females than males.

Neurofibroma

Definition

- It is a tumor like masses formed from nerve sheaths either neurolema (neurolemoma) of spinal nerves or schwannoma from cranial nerves usually the 8th nerve.
- It is considered as hamartoma.

Clinical Picture**→ FOR ALL TYPES:****Symptoms**

- Painless slowly growing swelling moves across and not along the course of the nerve.

O/E**► Inspection:**

- No. : **Solitary or multiple.**
- Size: **variable.**
- Shape: **rounded.**
- Site: **subcutaneous.**
- Cafe au lait patches: **brown patches seen all over the body.**

► Palpation:

- Edge: **well defined.**
- Consistency: **firm.**
- Mobility: **not attached to skin.**

→ ACCORDING TO DIFFERENT TYPES:**1- Generalized neurofibromatosis (Von Recklinghausen's disease):****➤ The most common type**

- Often it is familial
- No. : multiple
- Cafe au lait patches: especially on the back.
- Special characters: tender, ↑ ICT if arise from the intracranial nerve, no sensory or motor loss.
- Pheochromocytoma may be present (MEN IIb).

2- Solitary neurofibroma

- No.: single.
- Consistency: firm, tender.

3- Pachy dermatoceles (Plexiform Neuroma)

- Cystic swelling gives sensation as a bag of worms due to presence of multiple nerve fibers inside the swelling
- common in face & neck → facial deformity

4- Molluscum fibrosum

- Multiple soft fibrous swellings (cutaneous nerve endings are affected)
- Scalp, face and trunk are commonly affected (hand and feet escape).

5- Elephantiasis neurofibromatosis:

- Skin becomes thickened and replaced by grayish white glistening tissue (diffuse affection of the nerves of the skin and subcutaneous tissue).
- Commonly in children in limb.
- Hypertrophy of a huge part of a limb.
- Differentiated from lymphedema by congenital nature & café au lait patches.

6- Acoustic neuroma :

- May be associated with acoustic nerve tumor.
- It has a striking familial incidence showing itself as Mendelian dominant trait.
- It is often associated with tumors of the dura and choroid plexus.

Complications

- 1- Pressure symptoms e.g. deafness in acoustic neuroma.
- 2- Malignant transformation (neurofibrosarcoma).

D.D. (Multiple swellings)

1. Multiple neurofibromatosis.
2. Multiple lipomatosis.
3. Multiple melanoma
4. Multiple angioma.
5. Multiple lymph node enlargement.
6. Multiple warts.
7. Multiple exostosis (bony swellings).

Treatment

- Solitary neurofibroma → local excision
- Multiple neurofibroma → excision of the causing trouble.

Neurofibrosarcoma

- It is either de novo or on top of neurofibroma.
- Manifested by:
 - pain
 - rapid growth, hard in consistency
 - nerve paralysis.
- It may metastasize.

Treatment wide local excision or amputation.

Major Trauma

"Triage" "Poly-traumatized Patients"

Definition

- Trauma is body injury which is accompanied by local as well as systemic effects.

Incidence

- Represent the commonest cause of death among people aged 1-44 years.

Etiology

1- Closed (Blunt) trauma:

- Direct → e.g. motor car accident (most common), fall from height...etc.
- Indirect: fracture ribs.
- Spontaneous rupture.

2- Open (Penetrating) trauma: gunshot wounds, stab wounds, iatrogenic...etc.

- If a moving car stops suddenly, the person will continue moving forward (inertia).
- **Without a seat belt:** the head will strike against the car. However, the seat belt itself may cause injury to clavicle, GIT.
- **With a Seat belt:** may include fracture clavicle, trauma to the intestine or mesentery. The skin mark should raise suspicion.

Causes of death

- **Immediate (minutes):** Hemorrhage, associated injuries to vital structures.
- **Early (hours):** hemorrhage, major fractures.
- **Late (weeks):** sepsis, MOF.

Classification (TRIAGE SYSTEM)

- **Triage** involves sorting patients into three groups by colored labels according to their injury and availability of resources as follow:
 1. **Red:** those who will die anyway whether they receive medical attention or not.
 2. **Yellow:** those who will survive only if they receive timely medical attention. The first hour after injury is called **golden hour**.
 3. **Green:** those who will survive anyway whether they receive medical attention or not.

→ If the number of patients exceeds the resources, the yellow code is treated first.

→ If the number of patients doesn't exceed the resources, all codes are treated.

Management of Poly-Traumatized Patient

Pre-hospital management:(ABCDE)

- **Airway:** maintaining upper airways clear, even by insertion of cannula 14G in the cricothyroid membrane.
- **Breathing:** keep normal breathing rate by assisted or mouth to mouth breathing.
- **Circulation:** control of bleeding by compression
- **D** → Damaged limb → should be splinted.
→ Drugs → morphine if needed.

At hospital:

According to trauma life support system(ATLS)

⇒ 1st survey: (ABCDE)

A- Airway (and cervical spine control)

1. Clear airway.
2. Protect airway:
 - Oropharyngeal or nasopharyngeal airway prevents tongue from falling back & occluding the airway in an unconscious person.
 - Tracheal intubation is indicated with:
 - Apnea.
 - Risk of aspiration.
 - Impending or actual airway compromise (inhalation injuries, maxillofacial trauma).
 - Closed head injuries (allow hyperventilation to ↓ ICP).
 - Cricothyrotomy:
 - It is not suitable for children

Tracheostomy is rarely needed in the emergency room management

3. Cervical spine control:
 - Cervical spine immobilization is done using a backboard & a rigid collar.
 - If a collar is not available, **manual in-line immobilization** is necessary.

B- Breathing

1. Inspection: for air movement, respiratory rate, cyanosis, tracheal shift, jugular venous distension
 2. Palpation: for subcutaneous emphysema & flail segments.
 3. Auscultation for upper airway sounds (stridor, wheezing or gurgling).
 4. Percussion for hyperresonance or dullness over either lung field.
- **Action:** e.g. Needle decompression for tension pneumothorax.

C- Circulation

1. Control bleeding with direct pressure if possible.
2. Two large-calibre (16 gauge) peripheral IV lines are inserted.
3. Blood samples are sent for typing, cross matching, Hb%, HCT & blood chemistry.
4. IV fluids(ringer lactate) & blood are given.

D- Disability

- **AVPU evaluation:** based on patient's best response
 - **A:**Alert & interactive.
 - **V:**Vocal stimuli elicit a response.
 - **P:**Painful stimuli are necessary to elicit a response.
 - **U:**Unresponsive.
- Followed by Glasgow coma scale in the secondary survey.

E- Exposure & Environment

- **Clothes:** all clothes of the victim are removed using sharp large scissors.
- **Warmth:** keeping the emergency room warm & using blankets to prevent hypothermia.
- **Insert:**
 - Foley's catheter to monitor urine output (this is contraindicated if there is blood at the urethral meatus).
 - Nasogastric tube (Ryle's) decompresses the stomach & prevents vomiting & aspiration.
- Radiological assessment.

⇒ 2nd survey: (after stabilization of the patient general condition)

➤ This phase includes:

- 1- **Head to toe examination of undressed patient**
- 2- **Glasgow coma scale: in patients with head injury.**
- 3- **History: (AMPLE)**
 - Allergies.
 - Medications.
 - Past medical history.
 - Last meal (time).
 - Events of injury.
- 4- **Urgent investigations (after basic life support):**
 - **Laboratory:**
 - HB%, glucose level, Kidney functions, PO₂, PCO₂, Na⁺, K⁺
 - **Radiological:**
 - Plain X-ray: chest and skeletal or visceral injuries.
 - CT and MRI: chest, abdominal or head traumas.
 - U/S: for abdominal injuries.
 - Duplex for vascular injuries.
 - **Instrumental:**
 - Abdominocentesis or thoracocentesis.
 - GIT or urinary endoscopies.

A. Definitive Treatment

- According to the type of injury and priorities.

B. Rehabilitation

- Rehabilitation facilitates productive living for disabled people within the community.

Wounds

A - Definition: discontinuity of tissues produced by external trauma.

B - Types: 1. **Closed:** in which skin surface is intact.
2. **Open:** in which skin surface is disrupted.

C - Complications: general & local.

D - Treatment

Closed Wounds

A- Contusion:

- i. Ecchymotic bluish skin patches caused by blunt trauma
- ii. **Treatment** → fomentation (cold in 1st 24 hrs then hot after that)

B- Hematoma:

- i. Localized collection of blood
- ii. **Types:**
 1. Subcutaneous.
 2. Subperiosteal.
 3. Subfacial
 4. Intramuscular.
- iii. **Fate:**
 1. Organization by fibrosis.
 2. Infection (abscess formation).
 3. Calcification (myositis ossificans)
- iv. **Treatment:**
 - Antibiotics + pressure bondage to stop bleeding
 - Fomentation + Aspiration if large.

Open Wounds

A. Incised (Cut):

- Caused by sharp cutting objects like surgical wounds
- Little tissue damage with clean cut edges.
- Little or no infection.
- Severe bleeding due to clearly cutted vessels.

B. Lacerated:

- Caused by heavy blunt trauma.
- Severe tissue damage (more liable to infection).
- Little bleeding due to crushed vessels.

C. Degloving

- Skin and subcutaneous fat are stripped by avulsion from its underlying fascia, leaving underlying structures exposed

D. Bites:

- May be animal or human bites.

E. Gun shot wounds:

- May be high velocity missile injuries (rifles) or low velocity ones (pistols).

F. Abrasions:

- Partial injury of superficial layers of the skin due to friction against rough surface.

Treatment: antibiotics + Betadine antiseptic dressings.

Complication of Wounds

(I) General:

1. **Shock:** hypovolaemic, septic or neurogenic.
2. **Septicemia.**
3. **Crush syndrome:** (traumatic anuria).
 - ▶ Results from massive crushing of big muscles → myoglobin released in circulation → stuck in glomeruli → acute renal failure.
4. **compartmental syndrome (Acute limb ischemia).** As The crushed muscles swells inside its fascial covering → compartmental pressure → compression of vessels → acute limb ischemia.
 - ▶ **Treatment:** (of compartmental syndrome)
 1. **Medical**
 - a. Anti-shock measures.
 - b. Wash the kidney by strong diuretics (I.V mannitol).
 - c. Alkalinisation of urine by I.V sodium bicarbonate.
 2. **Surgical:**
 - Fasciotomy to prevent compartmental syndrome

(II) Local:

- ▶ **Infection:** pyogenic: staph, strept and pseudomonas
Specific: tetanus, gas gangrene.
- ▶ **Injury of:** vessels nerves, tendons, joints or solid organs
- ▶ **ischemia:** vascular or infective gangrene.
- ▶ **Complications of healing:**
 - Contractures & deformities.
 - Chronic ulcers.
 - Sinus formation.
 - Keloids.
 - Disfigurements.
 - Lymphoedema

Treatment of Wounds

Pre-hospital Management

1. Ensure patent airways if the patient is unconscious.
2. Ensure good breathing.
3. Compression by sterile dressing to prevent bleeding from the circulation.
4. **Drugs** as morphin 3 Anti (Antibiotics + Antitetanic serum + Anti-gasgangrene).
5. Stabilization of any orthopedic or vertebral fractures.

Hospital Management

ii. In the hospital the patient is resuscitated

iii. Rapid Examination & Investigations :

- To assess associated visceral, arterial, nerve, or bone injuries.
- (HB%, abdominal sonar or C.T scan, x-ray chest and bones for fractures, Duplex for vascular injury ... etc.)
- **For arterial injuries**
 - Feel the **distal pulses** , if not palpable reassess after treatment of shock.
 - Urgent duplex is mandatory if the pulses are not palpable

iv. Definitive treatment according to the priority

- ▶ Treatment of lacerated wound or crush injury.
 - **Irrigation of wound with saline.**
 - **Wound debridement:** removal of all fragments and devitalized tissues:

- Skin:

- **Clean (within 8 hours)** → closure without tension.
- **Infected (after 8 hours)** → leave it opened with sterile dressing then delayed closure or grafting.
- ▶ Clean & early wound (>6 hrs) → 1ry closure.
- ▶ Contaminated or late → delayed 1ry closure.
- ▶ All missile injuries require exploration.

➤ **N.B: In contaminated wounds:**

1. Don't do nerve repair.
2. Don't do tendon repair.
3. Don't close the deep fascia.
4. Don't close the skin.

Amputation may be rarely needed in:

- a-Severely crushed limbs b. Gangrene

Wound Healing

The wound healing includes:

- 1- Healing of wound.
- 2- Healing of bone.
- 3- Healing of nerve.

I- Wound Healing

*** It has 3 phases:**

***Phase I: Contraction:**

- This is mechanical reduction in the size of the defect.
- It starts 2 days after the injury and is complete within 2 week

***Phase II: Inflammation and granulation tissue formation:**

1. Inflammation stage. Lasts for about 5 days.

- During which there is ↑ vascularity + ↑ capillary permeability → exudation of RBCs + WBCs, plasma and fibrinogen and platelets → fibrin networks.
- ↑ Macrophages → ingestion of cellular debris and necrotic cells from the wound.

2. Granulation stage: Lasts about 2 weeks

- Proliferating fibroblasts + new capillaries → collagen fibers + ground substance (mucopolysaccharides and glycoproteins)
- Ascorbic acid (Vit. C) and ferrous iron enhance this stage.

3. Fibrous tissue stage

- Collagen fibers undergo remodeling into fibrous tissue (scar) which are arranged along the lines of tension of the wound.

***Phase III: Epithelial surface formation:**

A) Incised wound:

Basal cell layer of the epidermis starts migration over the fibrous tissue formed in the previous phase, to close the skin surface over the wound.

B) Large open wound:

Epidermis grows for few centimeters and then slow down → so better → cover by skin graft.

Types of wound healing:

- I. **Primary intention:** (occurs in clean incised wounds) when they are closed immediately by suture → minimal nice scar → neat scar.
- II. **Secondary intention:** (occurs in gaped wounds → with wound infection) more scar formation → ugly scar.
- III. **Tertiary intention:** left open for 5 days → will require a delayed primary suture after being clean

II- Bone Healing**1. Stage of Fracture hematoma & granulation tissue (2 weeks)****➤ Characterized by:**

- Capillary growth in the hematoma site
- Phagocytic cells invading and cleaning-up debris in injury site
- Fibroblasts migration
- Vasodilators: {e.g. histamine due to soft tissue injury} bring more proteins & minerals
- Acidic in reaction: this dissolves Ca from bone ends → so, concentration of Ca in fracture hematoma
- Phosphatase enzyme: produced by osteoblasts → split the organic bound Ca → so help in saturation of fracture hematoma by Ca

2. Stage of Primary Callus (2 months)

- The granulation tissue is gradually replaced by cartilage.
- It's alkaline to help precipitation of Ca salts.

3. Stage of Bony Callus

- Continued migration and multiplying of osteoblasts and osteocytes results in the fibrocartilaginous callus turning into a bony callus.

4. Remodeling stage

- Internal & external callus is removed and compact bone is laid down in intermediate callus in order to reconstruct the shaft.

III- Nerve Healing

Neuropraxia	Axonotmesis	Neurotmesis
- Nerve concussion: physiological interruption of all its functions without any organic damage.	- Rupture of nerve fibers but outer sheath is intact	- Division of the nerve (partial or complete).
- Complete motor paralysis.	- Complete motor paralysis.	- Complete motor paralysis.
- Patchy sensory loss.	- Complete sensory loss.	- Complete sensory loss.
- No Wallerian degeneration.	- Wallerian degeneration (2 wks) followed by - Regeneration (1 mm/d).	- Wallerian degeneration occurs but regeneration is impossible.

Factors affecting healing

A) General Factors

1- **Type of patient:**

old patients have poor wound healing d.t. reduced rate of protein turnover.

2- **Medical disease:** DM, LCF, uremia, CRF lead to poor wound healing

3- **Malnutrition.**

- Protein deficiency leads to diminished synthesis of collagen & ground substance.
- Vitamin A deficiency leads to deficient epithelization.
- Calcium, Zinc, Copper & Manganese also affect wound healing.

4- **Medications e.g. steroids,** inhibit wound healing.

5- **Smoking.**

6- **Irradiation:** leads to E.A.O. of wound edges.

B- Local Factors

1- **Vascularity:** good blood supply (e.g. in the face & scalp) leads to good healing while poor blood supply (wounds below the knee) causes delayed healing.

2- **Mobilization:** early mobility delay wound healing.

3- Increased tension: by tight sutures or haematoma lead to ischemia of wound edges.

4- **Infection:** bacteria compete with fibroblasts for O₂ & nutrition, also bacteria secrete collagenolytic enzymes, which destroy collagen fibers.

5- **Foreign bodies & necrotic tissue:** impair wound healing.

6- **Adhesion of the wound to a bony surface:** (e.g. wounds over the shin of the tibia) prevents wound contraction → impairs wound healing.

7- **Denervation:** (e.g. peripheral neurotherapy) interfere with proper healing.

Surgical site infection (SSI)

Definition

- They are non-specific infections following wounds.

Etiology

➤ **Predisposing factor:**

- General patient characteristics: age, malnutrition, etc.....
- Wound characteristics: FBs, tissue ischemia, etc.....
- Operative characteristics: poor surgical techniques (the most important factor),

➤ **Source of infection:**

- Endogenous: human body flora.
- Exogenous: from surgical team, instruments, dressing etc

➤ **Causative organisms:**

- Commonly ***Staphylococcus aureus*/MRSA, *Streptococcus pyogenes*, *Enterococci* and *Pseudomonas aeruginosa***

Classification

A. **Clean wounds (class I):**

- Elective non-traumatic wounds in which the gastrointestinal, urinary or respiratory tracts are not entered.
- Risk of SSI → less than 2%.

B. Clean contaminated (class II):

- Elective surgery into the gastrointestinal, urinary or respiratory tracts with no significant spillage.
- Risk of infection → 2-5%.

C. Contaminated wounds (class III):

- Open accidental wounds encountered within 4 hours.
- Gross spillage from GIT.
- Incision through non-inflamed, non-purulent tissues.
- Risk of infection → 10-20%.

D. Dirty wounds (class IV):

- Traumatic wounds more than 4 hours.
- Purulent infection or necrotizing soft tissue infection.
- Perforated viscus accompanied by high degree of contamination.
- Risk of infection → up to 40%.

Clinical Picture

Usually appears between the 5th & 10th day postoperative.

➤ Symptoms:

- General: fever, anorexia, headache & malaise.
- Local: - Pain - Swelling
 - Disturbance of function: The wound hasn't healed within 10 days after the injury

➤ Signs:

- General: fever & tachycardia
- Local:
 - The wound show: redness, tenderness, Pus or cloudy fluid is draining from the wound & pimple or yellow crust has formed on the wound.
 - Fluctuant areas and crepitus may be felt.
 - LN: large and tender.

Complications of surgical infections:

1. Spread of infection:
 - a. Direct
 - i. Necrotizing infections
 - ii. Abscesses
 - iii. Phlegmons. These contain little pus but much oedema
 - b. Lymphatic: especially streptococcal infection
 - c. Blood.
2. Fistulae and sinuses
3. Necrosis or gangrene of the affected part
4. Suppressed wound healing
5. Immunosuppression and superinfection
6. Systemic inflammatory response syndrome (SIRS) and Multiple organ system failure (MOSF): the most serious complication

Investigations

- Laboratory: CBC: ↑TLC
- Microbiology: C&S.

Treatment

Prophylaxis

The patient

- Correct any PDFs as control of diabetes, stopping of smoking and correction of nutritional deficiency.
- Skin preparation with appropriate antiseptics.

The surgeon

- Meticulous surgical technique by proper hemostasis, gentle handling of tissue & avoiding tight sutures or leaving dead space.

Prophylactic antibiotics

➤ Clean operations:

- No need for prophylactic antibiotics except in:
 - When an implant, e.g. prolene mesh or a vascular graft is used.
 - In patients with valvular heart disease to prevent infective endocarditis.
 - In emergency surgery in a patient with preexisting active infection.
 - When infection would be very severe or have life-threatening consequences as in aortic surgery or organ transplantation.

⚡ Recommendation:

- One dose of 1st generation cephalosporin, or (ampicillin + sulbactam).

➤ Clean contaminated operations:

- One dose of 2nd generation cephalosporine or (ampicillin + sulbactam) + aminoglycoside.

➤ Contaminated operations:

- As contaminated, but add metronidazole.
- In all if the operation exceeds 2 hrs, another dose is prescribed.

Treatment

- Liberal drainage: The wound should be opened by removal of skin stitches.
- Antibiotics are used in invasive infections guided by culture & sensitivity tests.
- Possible sources of hospital acquired infection should be traced and corrected.

Blood Transfusion

Definition

- The process of transferring of blood or blood-based products from one person into circulatory system of another.

Indications

A. To restore blood volume

1. Acute hemorrhage external or internal.
2. Operative replacement.
3. Post operative replacement.
4. Severe burn.
5. Strangulation.
6. Exchange transfusion in case of Erythroblastosis fetalis

B. To provide deficient blood element

1. Packed RBCs → severe anemia.
2. WBCs → severe leucopenia & agranulocytosis.
3. Fresh frozen plasma → Hemophilia, Liver cell failure, Burn, Crush \$, Malnutrition.
4. Platelets concentrate → acute leukemia, Thrombocytopenia.
5. Cryoprecipitate → hemophilia, Von willibrand disease & DIC
6. Albumen → hypoalbuminaemia.
7. Clotting factor conc. → hemophilia, Von willibrand disease.
8. Fibrinogen → hypofibrinogenemia & DIC.

Test the blood for

- ABO antigen.
- Rhesus antigen.
- Direct, indirect Coomb's test.

Blood storage

- 120 ml of ACD + 450 ml blood.
- Kept at 4°C.

Complication

Acute (within 1st 72 hrs)

Immunological

1-Hemolytic reaction:

E/O: Due to incompatible blood transfusion (usually d.t. human error)
→ destruction of donor RBCs by the recipient Ab.

Clinical picture:

→ In conscious patient

Symptoms	Signs
1. Fever & rigors	1. Fever & rigors.
2. Chest pain.	2. Tachycardia.
3. Dyspnea.	3. Cyanosis.
4. Headache.	4. Hypotension.
5. Lumber pain.	5. Oliguria.
6. Nausea.	6. Later on renal failure.
7. Vomiting.	

→ If patient is under anesthesia or comatosed

Incompatibility is suspected by:

1. Bleeding tendency (oozing of blood) → **the most important sign.**
2. Progressive unexplained hypotension.
3. Unexplained tachycardia.

Treatment of haemolytic reaction

1. Stop the transfusion immediately.
2. IV fluid (ringer lactate + corticosteroid).
3. Alkalinization of urine by 8.4% NaHCO₃ (40 ml).
4. Mannitol 20% 100 ml (**forced alkaline diuresis**).
5. Repeat patient's blood typing & matching.

2-Febrile reaction:

E/O: minor bacterial contamination.

C/P: chills, fever, N&V & headache.

TTT: Stop the transfusion + antipyretic.

3-Allergic reaction:

C/P: Ranging from urticarial patches up to laryngeal edema.

TTT: - Antihistaminics & cortisone.

- If reaction is severe → stop the transfusion.

Non-Immunological

1-Complication of massive blood transfusion

- Acidosis.
- Hyperkalemia.
- Circulatory overload (heart failure).
- Hypothermia.
- Citrate toxicity.
- Bleeding tendency due to:
 - Dilution of the patient blood platelets.
 - Deficient clotting factors.
 - Activation or ↑ production of fibrinolysin.
 - Citrate toxicity.

2-Air embolism.

3-Thrombophlebitis at the site of injection.

4-Complication of transfusion of stored blood

- Acidosis. - Hyperkalemia. - ↑ O₂ affinity

Delayed (after 72 hrs)

I-Immunological

a-Delayed hemolysis

b-Post-transfusion purpura

II-Non-Immunological:

a-Iron overload (hemochromatosis).

b-Transmission of diseases as

- 1) AIDS.
- 2) Brucellosis.
- 3) CMV (the commonest).
- 4) Malaria (only by RBCs).
- 5) Viral hepatitis.
- 6) Syphilis.

Complications in the donor

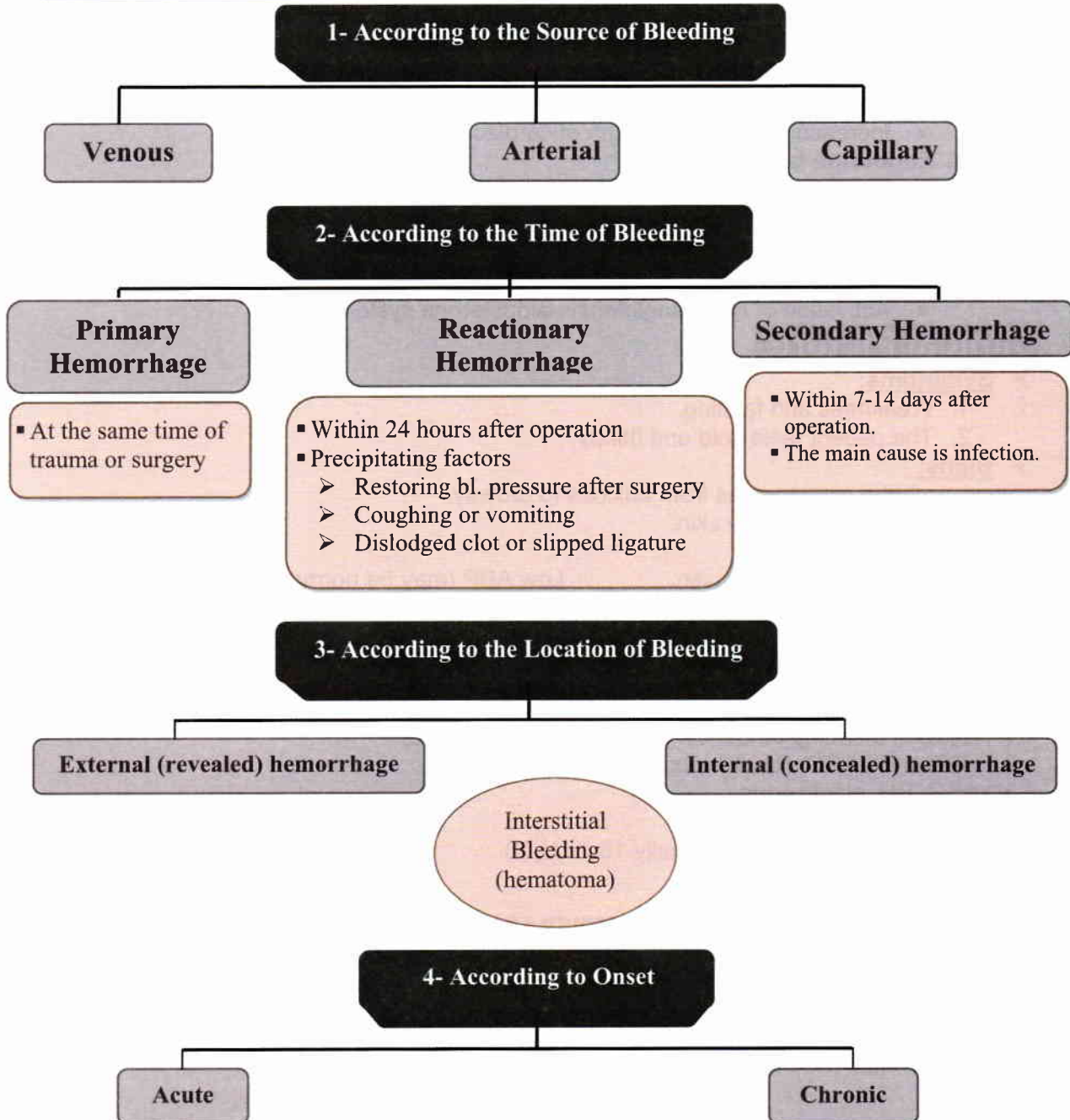
- ❖ Anemia.
- ❖ Complication of repeated venous puncture

Hemorrhage

Definition

- Loss of circulating blood from damaged blood vessels.

Classification of Hemorrhage



Physiological response to hemorrhage

1. Stopping the bleeding

- Vasoconstriction and retraction of the intima
- Platelet plug.
- Blood clotting.

2. Maintaining the effective circulatory volume → To maintain perfusion of brain and heart at expense of muscles, skin and viscera.

A. Neural factors: due to sympathetic stimulation

- Constriction of veins → shifts blood to the arterial circulation.
- Constriction of arterioles → increases peripheral resistance.
- Increased rate and strength of cardiac contraction

B. Endocrine factors:

- Release of catecholamine → increases heart rate and contractility, and cause constriction of the arterioles of the skin, kidney and viscera
- Release of stress hormones e. g. cortisol, glucagon...etc.
- Release of ADH
- Activation of renin-angiotensin-aldosterone system.

Clinical Picture

➤ Symptoms:

1. Weakness and fainting.
2. The patient feels cold and thirsty.

➤ Signs:

- mental status:varies from anxious to drowsy.
- Pale, cold, clammy skin.
- Vital signs:
 - Rapid weak pulse.
 - Low ABP (may be normal then ↓)
 - Subnormal temperature.
 - Rapid deep respiration.
- Oliguria & later on renal failure.

Investigations

(It's an emergency condition, investigations are part of resuscitation)

1. Exclude bleeding tendency: BT, PT, APTT and CBC.
2. HB%, Hct, ABO, Rh → blood transfusion.
3. ABG, PH, electrolytes.
4. KFTs & LFTs.
5. Measurement of CVP (normally 15cm.H₂O).

Treatment

1. Stop hemorrhage: (position - pressure - packing) e.g.

- **Elevation of the limb** above the heart level stops venous bleeding and decreases arterial bleeding.
- **Pneumatic anti-shock garment (PASG)** → tamponade lower limb, pelvic and abdominal hemorrhage.
- **Balloon tamponade** for hemorrhage from esophageal varices.

2. **Restore blood volume:** through a wide bore cannula or a cut down on the long saphenous vein, if necessary. The volume depends on the class of hemorrhage:
 - ➔ **Class I:** no replacement.
 - ➔ **Class II:**
 - **Estimation of deficit:** 15-30% (750-1500 ml/70 kg).
 - **Type of fluid:** Ringer lactate.
 - **Volume of replacement** = 3 times the estimated deficit ($\approx 3L$).
 - ➔ **Class III and IV:**
 - **Estimation of deficit:** $> 30\%$ (> 1500 ml).
 - **Type of fluid:** Ringer lactate+blood transfusion.
 - **Volume of replacement** = estimated deficit ($\approx 1500-2000$ ml).
 - **Administration:** continue until the hematocrit reaches 30%, the urine output is 50 ml/hour, and the CVP rises to the upper half of the normal range.
3. **Optimize oxygen delivery:** 40% oxygen is given for class II hemorrhage and 100% for classes III and IV.
4. **General care of the patient:** absolute bed rest and analgesia (morphia, 3-5 mg IV), repeated if necessary. Morphine is contraindicated in head injury and in cases of respiratory and liver insufficiency.
5. **Monitoring:**
 - Urine output, core temperature, hematocrit and cardiac monitoring (ECG for early detection of shock-induced arrhythmias is important).
 - In class III or IV hemorrhage $\rightarrow$ as above + central venous pressure (CVP), blood gases and pH.

Shock

Definition

- A state of peripheral circulatory failure and impaired tissue perfusion resulting in disturbed cellular metabolism

Classification and etiology

- 1- Hypovolemic:d.t. $\downarrow$ blood volume which may be d.t. blood loss(internal or external he), plasma loss(burns), fluid loss(severe vomiting or diarrhea).
- 2- Cardiogenic:d.t insufficient myocardial function e.g. extensive myocardial infarction.
- 3- Neurogenic:d.t. vasovagal attack(severe pain) or spinal shock which lead to peripheral vasodilatation $\rightarrow$ peripheral pooling of blood.
- 4- Anaphylactic shock: d.t antigen-antibody reaction $\rightarrow$ severe V.D. & peripheral pooling of blood.e.g. injection of penicillin or antitetanic serum.
- 5- Septic: commonest cause is Gram -ve bacilli through genitourinary tract.
- 6- Endocrinal: e.g. adrenocortical insufficiency.

Pathophysiology

A. Cellular:

- Tissue hypoperfusion $\rightarrow \downarrow O_2$ delivery to the tissues $\rightarrow$ anaerobic metabolism $\rightarrow \uparrow$ production of lactic acid $\rightarrow$ metabolic acidosis.

B. Microcirculatory changes:

- Under the effect of catecholamines the precapillary sphincters constrict leading to:
 - Further slowing of the capillary circulation called **slugging** of blood.
 - **Slugging** encourages spontaneous coagulation of blood in these capillaries

- If extensive, it is called **disseminated intravascular coagulation (DIC)**.
- This depletes coagulation factors and induces bleeding in the rest of the body (consumption coagulopathy).

C. Systemic:

a) Adrenal response:

- Release of catecholamine → causes of redistribution of blood, diversion of blood from non-vital organs (skin, GIT) by V.C. to vital organs by V.P. (coronary Vs) & Ms
→ Increases heart rate and contractility.
- Release of stress hormones e. g. cortisol, glucagon...etc.
- Release of ADH
- Activation of renin-angiotensin-aldosterone system → vasoconstriction – Na⁺ & water retention.

b) Neural response: due to sympathetic stimulation

- VC of veins → ↑ venous return.
- VC of arterioles → ↑ peripheral resistance.
- ↑ rate and strength of cardiac contraction.

c) Individual organs

- 1- Brain: irritability and loss of consciousness later on.
- 2- Heart: hydropic degeneration and failure.
- 3- Lungs: ARDS.
- 4- Kidney: reversible renal tubular necrosis then irreversible cortical necrosis.
- 5- Blood: DIC.
- 6- Muscle: lactic acidosis.
- 7- Intestine: VC with bacterial translocation ending in septic shock.

Clinical Picture

A- C/P of shock:

➤ Symptoms:

- a. Weakness and fainting.
- b. The patient feels cold and thirsty.

➤ Signs:

- Mental status: varies from anxious to drowsy.
- Pale, cold, clammy skin.
- Vital signs:
 - Rapid weak pulse.
 - Low ABP (may be normal then ↓)
 - Subnormal temperature.
 - Rapid deep respiration.
- Oliguria & later on renal failure.

C/P of septic shock is divided into 2 phases;

- 1- Hyperdynamic phase: Flushed warm skin with fever, mild ↓ ABP with tachycardia and tachypnea and high cardiac output heart failure.
- 2- Hypodynamic stage: Cold clammy skin and marked ↓ ABP.

B- C/P of the cause:

- Sites of bleeding or internal hemorrhage in hypovolemic shock.
- Genitourinary or chest infection in septic shock.

C- C/P of complications: Multiple organ failure e.g. anuria and ARDS.

Investigations (Urgent)

- Blood sample: ABO typing, ABG and Hb %
- For the cause: ECG, U/S, CT scan
- For complications: KFTs, ABG, LFTs, Hb%

Treatment**I- Treatment of shock:**

Immediate intervention is mandatory

A- First Aid(prehospital manag.):

- Airway: should be patent. - Breathing: is maintained.
- Circulation: stop any external bleeding by compression
- Drugs: morphia for pain.
- 3 Anti: antibiotics, anti gas gangrene, anti-tetanic serum
- Elevation of legs - Fractures are immobilized. - Warmth.

B- AT hospital

- Resuscitation and primary surgery:
 - Lines: 2 cannula for fluid replacement and sampling.
 - Ryle: to evacuate stomach.
 - Catheter: to check urine output.
 - O2 inhalation: to ↑ O2 content of blood
- Fluid administration: which may be
 - Crystalloids: as saline, ringer or ringer lactate (dextrose 5% not used in shock).
 - Colloids: as dextrans (high or low molecular weight) or plasma.
 - Blood: given in severe blood loss (acc. To blood typing & cross matching).
- Inotropic drugs: as dopamine & dobutamine → improve the myocardial contractility
- Monitoring:
 - Pulse, ABP, Urine output, temperature. - Arterial line for ABG.
 - Central venous pressure catheter for volume replacement and to avoid fluid overload.
 - Swan-ganz catheter: may be needed in left ventricular disease.

In Septic shock: we must

- 1- Eradicate sepsis.
- 2- Give parenteral, broad spectrum antibiotic.

II- Treatment of the cause

- Bleeding: exploration and repair. - Burns: fluid replacement and wound care.
- Sepsis: eradication of septic focus by drainage and antibiotics.

III- Treatment of complications

- e.g. DIC: fresh frozen plasma.
- Follow-up should be maintained till the patient restores normal physiology.

Prognosis

- The worst prognosis accompanying septic shock which carry high mortality rate

NB Hypovolemic and septic shock see the basic book

Hypokalemia

Definition

- Serum K level < 3.5 mEq/L.

Etiology

- 1- Decrease intake: starvation.
- 2- Decrease absorption: malabsorption syndrome.
- 3- Increase intracellular content:
 - Insulin therapy. - Alkalosis
 - Beta-2 stimulation: adrenaline therapy, bronchodilators, stress as MI.
 - Increase cellular synthesis: during treatment of megaloblastic anemia.
 - Familial hypokalemic periodic paralysis.
- 4- Increased renal loss:
 - Diuretics: loop or thiazides.
 - Increased aldosterone: liver cell failure, congestive heart failure, nephritic syndrome, Conn's syndrome, corticosteroid therapy and liquorice.
 - Renal tubular dysfunction: drugs (aminoglycosides, cytotoxic drugs)
- 5- Increased GIT loss: diarrhea, vomiting, use of gastric suction, external GIT fistula.

Hazards

- 1- ↓↓ nerve and muscle excitability.
- 2- ↑↑ risk of supraventricular arrhythmias in patients taking digoxin.
- 3- ↑↑ risk of hepatic coma in patients with liver disease.
- 4- Polyurea due to ↓ responsiveness to antidiuretic hormone.

Clinical Picture

- 1- Asymptomatic in mild cases.
- 2- Skeletal muscles: weakness up to paralysis.
- 3- GIT: constipation up to ileus.
- 4- Cardiac: arrhythmias especially in patients with myocardial ischemia or in those who taking digitalis.
- 5- Glucose intolerance.
- 6- Nephrogenic diabetes insipidus.
- 7- Clinical picture of the cause.

Investigations

- 1- Low serum potassium.
- 2- ECG changes.
- 3- Urinary potassium level.
- 4- Investigations of the cause.

Treatment

- 1- Treatment of the cause:
- 2- Potassium replacement:
 - **Oral potassium** can be used for mild cases if there no life-threatening symptoms.
 - **IV potassium** should be given slowly in a large vein with continuous ECG monitoring, urine output at least 40 ml/hour, no more than 40 mEq/added to 1 L fluid and no faster infusion than 40 mEq/Hour.
 - Estimation of potassium deficient = deficient X 1/3 body weight (1/2 correction)

Hyperkalemia

Definition

- Serum K level > 5.3 mEq/L.

Etiology

1- Pseudohyperkalemia:

- Increased release from damaged cell as in hemolysis or leukemia.
- Increased release from damaged muscles as in vagus fist clinching or tourniquet application.

2- Increased K intake:

- During correction of hypokalemia. - Transfusion of old or irradiated blood.

3- Increase release from the cells:

- Insulin deficiency. - Metabolic acidosis.
- Cellular damage: tumor lysis syndrome. - Digitalis toxicity.
- Hyperkalemic periodic paralysis.

4- Decreased excretion:

- Renal failure. - Aldosterone antagonist: spironolactone.
- Hypoaldosteronism: Addison's disease, drugs (ACEI).

Clinical picture

- 1- Skeletal muscle weakness up to paralysis.
- 2- Intestinal hypotonia leading to constipation up to ileus.
- 3- Cardiac: bradycardia up to cardiac arrest.
- 4- Apathy and confusion.

Investigations

- 1- Serum K level.
- 2- ECG changes.
- 3- Urinary potassium.
- 4- Investigations for the cause.

Treatment

- 1- Treatment of the cause.
- 2- Correction of hyperkalemia:
 - If K > 7 mEq/L → dialysis.
 - If K < 7 mEq/L → IV sodium bicarbonate 100 ml IV infusion.
 - glucose 25% 100 cc + 20 unit regular insulin infusion over 3 hours.
 - Beta-2 stimulation by injection or inhalation.
 - IV Ca⁺⁺ gluconate as calcium antagonize K effect on the heart

Surgical Infections

	Tetanus	Gas gangrene
Definition	Acute specific infection lead to ↑ nervous excitation due to release of neurotoxin	Acute specific infection lead to spread of gangrene with excess gas formation
Etiology		
Organism	Clostridium tetani: Gm +ve anaerobic spore-forming bacilli giving drum stick app.	Gram +ve spore-forming bacilli Saccharolytic and proteolytic groups
Predisposing factors	-↑ organism content (contamination in fields and streets) -↓ O ₂ content: deep lacerated wounds, hemorrhage and shock. -Lack of proper sterilization of cat gut and instruments.	
Route	Wounds: especially occurred in fields or streets.	
	- Post-operative tetanus. - Tetanus neonatorum.	-endogenous infection.
Pathology	General: The organism release exotoxin act as: 1- Anti-choline esterase → tonic rigidity. 2- Extreme excitability of motor neurons → clonic contraction. Local: Minimal inflammatory reaction	Local: Sacchrolytic group act on CHO of dead muscle → gases → elevate sacrolemma → cut blood supply. Proteolytic: act on proteins → ammonia → H₂S H ₂ S + iron of Hb → iron sulphide black color and bad odor General: RBCs hemolysis, degenerative changes
Clinical picture IP	1-21 days	1-2 days
Symptoms	General: FAHM Local: - Pain at the site of wound. - Swelling: minimal - Disturbed function → convulsions	General: FAHM but fever is minimal Local: Pain: marked then disappears. Swelling: black offensive Disturbance of function → paralysis of affected limb.
Signs	General: 1- Stage of tonic contraction e.g trismus (earliest), risus sardonicus, dysphagia, dyspnea and stridor. 2- Stage of clonic contraction: clonic spasm on the tonic spastic muscle occur with excitation as light or noise Local: red, hot, tender wound	General: Tachycardia: earliest sign Jaundice, MOF Local: wound crepitus, foul odor, black color & serosanguinous watery discharge. No muscle contraction nor bleed on incision

Complications	- Asphyxia - Exhaustion - Respiratory and heart failure - hyperpyrexia	Severe toxemia and MOF
DD	Clonic contraction: - Strychnine poisoning. - Hydrophobia in rabies Tonic contraction - Tetany. - Trismus. - Meningitis.	Infection with gas forming organism Surgical emphysema
Investigations	-TLC:leukocytosis. -Smears from wound	- TLC: leucopenia and anemia ↑ bilirubin - Smear from discharge - Plain-X-ray: tissue gases.
Treatment	A- Proper management of the wound: - Skin is insed&deep fascia is opened. - Excision of dead muscles - Wash the wound with H₂O₂ - If less than 8hrs→close the skin loosely without deep fascia. - If more than 8hrs→leave the wound opened.	
Prophylactic	B- General: Sterile instruments and sutures	B- General: -Sterile instruments and sutures -Hospital us closed and sterilized in nosocomial infection
	C- Specific: <u>Immuni patient</u> -Clean wound → nothing Tetanus prone → toxoid + antibiotics <u>Non-immunized</u> Clean: TIG + immunization Tetanus prone: TIG + toxoid + immunization + antibiotics <u>Uncertain immune:</u> As non-immune	C- Specific - Polyvalent anti-gas gangrene serum - Penicillin
Curative	General -Resuscitation and monitoring in quite room. -O₂ or tracheostomy for cyanosis. Specific: Anti-tetanic Ig Crystalline penicillin Others e.g. Control convulsions by valium or muscle relaxant Cooling for hyperpyrexia	General: -R&M in isolated place -Hyperparic O₂ -blood transfusion&IV fluids Specific: Anti-gas gangrene Ig Crystalline penicillin Others Amputation above all affected muscles
Prognosis	Bad prognosis in short IP, unimmunized and extremes of age	Varies according to extent and severity of injury and time of TTT.

Acute Abscess

Definition

- A localized suppurative inflammation.

Predisposing Factors

- Poor general resistance, Bad hygiene

Route of Infection

1. **Direct** → through a wound or ulcer.
2. **Blood** → from a septic focus (bacteraemia).
3. **Lymphatic** → from an infected area.

Organism

- Usually staph, less commonly strep and E. coli.

Pathology

1. Central zone:

- Liquifaction, by dead WBCs.
- Pus is formed of:
necrotic tissue, inflammatory exudates,
organism, dead leucocytes.

2. Intermediate zone: formed of granulation tissue (pyogenic membrane)

3. Peripheral zone: of hyperemia

Complications & Fate

Generally:

- **Toxemia** → bacterial toxins circulating in the blood.
- **Bacteremia** → Transient presence of bacteria into the blood.
- **Septicaemia** → Persistent bacteremia multiplying in the blood.
- **Pyemia** → septic emboli from infected thrombus

Locally:

- **Pointing & rupture** (commonest sequelae) along the line of least resistance.
- **Chronicity** due to:
 - Organism tend to be chronic
 - Persistence of predisposing factors
 - Inadequate drainage & treatment.
- **Spread:** cellulites, lymphangitis and lymphadenitis.
- **Antibioma:** if sterile pus is formed and not drained.
- **Sinus or fistula formation.**

Clinical Picture

⇒ Symptoms:

General (FAHM)

Local

- Pain: throbbing & relived by elevation of the part.
- Swelling: edema
- Loss of function.

⇒ Signs:

General: fever, tachycardia.

Local

- Swelling → Hot & red, Tender
→ after 2-3 days pus points & rupture
- LNs → Enlarged, Elastic, Tender, Mobile
- Fluctuation is very late

Treatment

⇒ **General:** Analgesics, antibiotic, antipyretic (**AAA**)

⇒ **Local:**

1. **Rest of the affected organ**
2. **Hot fomentation.**
3. **Incision & drainage**

A- Classic method:

- Under general anesthesia (local anesthesia does not act in presence of infection).
- The incision should be:
 - Long & Dependent
 - Never cross a skin crease
 - Parallel to nerves & vessels
- Introduce a finger to break all septa.
- Packing for 48 hours for hemostasis
- later on dressing without packing

B- Special methods:**i- Hilton's method:**

- Used to avoid injury of important structures.
- The skin is only incised
- Abscess cavity is entered by closed blades of artery forceps
- Then the blades are opened widely.

ii- Aspiration used in

- Amebic liver abscess
- Brain abscess
- Cold abscess (Z technique).

NB. Chronic abscess:

- Thin walled → incision & drainage
- Thick walled → excision

	Cellulitis	Erysipelas
Definition	invasive non-suppurative infection of loose C.T	Non suppurative diffuse sharply demarcated streptococcal infection of superficial lymphatic vessels
Organism	usually strept	Usually strept
PF & Route	see before	
Complication	General + Chronicity + Pus locus +post-streptococcal G.N.+SIRS	
C/P	Symptoms → see before Signs <u>General</u> → see before <u>Local</u> tender, red, hot area with ill defined edge never affect the ear pinna.	Symptoms → see before Signs <u>General</u> → see before • <u>Local</u> the picture is similar to cellulitis but with some differences: 1. The skin is rose pink. 2. The edge is well defined ,slightly raised and often shows minute vesicles just beyond the spreading margin. 3. There may be islets of inflammation beyond the spreading margin separated from the main area by apparently normal skin.
TTT	See before (AAA + rest + hot fomentations)	

	<u>Carbuncle</u> (infective gangrene of the nape)	<u>Boil</u> (Furuncle)
Definition	Acute staphylococcal infection of S.C. tissues → Infective gangrene	Acute staphylococcal infection of hair follicles → perifolliculitis
Organism	Usually Staph aureus	
PF & Route	see before	
Pathology	Sites: Nape of neck (commonest), back, face Pathogenesis → Infection in hair follicles → spreads to underlying SC tissue → separate into loculated abscesses → each abscess burst on surface by a separate opening	Sites: → Hairy areas Pathogenesis → (perifolliculitis – central necrosis & suppuration) forming red, tender, hot area with a hair coming out from it.
Complications	1. General + Chronicity & Spread 2. Cavernous sinus thrombosis: if manipulated in dangerous area of the face. 3. Blind boil	

		<u>Carbuncle</u> (infective gangrene of the nape)	<u>Boil</u> (Furuncle)
C/P	Symptoms	→ see before	
	Signs	<u>General</u> → see before <u>Local</u> swelling → Hot & red, Tender → at first indurated then soften at center.	
TTT		→ after 2-3days multiple pustule appear on surface → bursting → cribriform app.	→ pus points & ruptures
		1. AAA + rest + hot fomentations 2. <u>Improve general condition</u> 3. <u>Once pus is formed</u> - Cruciate incision to open all pockets. - Debridement of necrotic tissues. - Dressing until healthy tissue is formed. - Leave to heal or use skin graft (the best).	3. Hot fomentation. 4. Incision & Drainage.

Ludwig's Angina

Definition

- Diffuse cellulitis of the floor of the mouth.

Organism

- Mixed infection (mainly streptococci).

Route of Infection

- Direct spread from infected lower teeth, tongue, mandible or salivary glands.

Clinical Picture

➤ Symptoms

- General: FAHM
- Local: dyspnea and dysphagia (edema of the floor of the mouth elevates the tongue)

➤ Signs:

- General: fever and tachycardia.
- Local: swelling of the floor of the mouth pushing the tongue up and backward

Complications

- Spread of infection to larynx → laryngeal edema.

Treatment

- **Early:** massive antibiotic (penicillin)+rest in a semi-sitting position to avoid airway obstruction.
- **Late:** submental release incision of skin and deep fascia.

Hand Infections**General Principles****Predisposing Factors**

- Bad hygiene, poor general condition
- More in manual workers

Organism

- Staph (90%), Strept, E. coli

Route of Infection

- Usually direct, blood-borne is rare.

Clinical Picture**Symptoms**

General → FAHM

Local

- Pain → dull aching then become throbbing when pus is formed
- Swelling → due to edema of the tissue.
- Disturbance of function → Limited movement

Signs

General → fever, tachycardia

Local → diffuse edema maximum on the dorsum of hand +L.Ns are enlarged, elastic & tender.

Complications

- **General:** toxemia, bacteremia, septicemia or pyemia.
- **Local:** chronicity, spread, pointing and rupture.

Treatment**A. Before suppuration**

General → Antibiotic, analgesic and antipyretic(AAA).

Local → (HHH)

1. Hot fomentation.
2. Hand elevation (to diminish pain and edema) by
Arm to neck sling(in minor cases)
Or Elevate the hand above level of
the body(in major cases).
3. Hand position
 - If resolution is expected → put the hand in position of rest.
 - If stiffness is expected → put the hand in position of function.\

1. Under general anesthesia.

2. **Bloodless field by** → limb elevation for 2 minutes
→ applying sphgmomanometer calf inflated
40mmHg systole
3. **Incision**at the site of selection, never cross skin crease.
4. **All pus & granulation tissues** are removed.
5. **No drain is allowed but a piece of tulle grass**
might be used.

Pulp space infection

Anatomical view

- It represents sc tissue related to the palmar surface of the distal phalanx
- It contains fat, fibrous septa and digital artery which gives epiphyseal branch before entering the space

Definition

- Infection of sc compartment related to the palmar surface of distal phalanx

Incidence

- 2nd common hand infection.

Etiology

- **Organism:** staph, strept and E.coli
- **Route**
 - 1- Direct: commonest
 - 2- Blood: rare
- **Predisposing factors:**
 - More in manual workers
 - Bad hygiene and bad general health

Pathology

- **Type:** suppurative inflammation
- **Gross picture:** hyperemia and edema
- **Micro:** acute inflammatory cells

Clinical picture

⇒ Symptoms

- **General:** FAHM
- **Local:**
 - Pain: dull aching → throbbing
 - Swelling: of distal phalanx
 - Disturbance of function: limited movement of distal phalanx

⇒ Signs

- **General:** fever and tachycardia
- **Local:**
 - Hotness, redness, tenderness and edema at dorsum of hand and 1st indurated then cystic
 - LNs: enlarged, elastic, tender and mobile

Complications

⇒ General

- Bacteremia, toxemia, septicemia and pyemia

⇒ **Local:**

- Chronicity, spread and abscess formation
- Thrombosis in digital arteries → osteomyelitis except epiphysis → parrot peak deformity.

Investigations

- Laboratory: CBC, C & S
- Radiological: plain x-ray for sequestration

Treatment

⇒ **Prophylactic: avoid predisposing factors**

⇒ **Therapeutic: as before but**

- Incision: anterolateral on the lateral side of distal $\frac{2}{3}$ of distal phalanx
- Counter incision in severe cases
- Hook stick for sequesterectomy

Prognosis

- Depends on time of intervention and severity of infection

Acute Paronychia

Definition

- Infection of tissue surrounding nail fold

Incidence

- Commonest hand infection

Etiology

- **Organism:** as pulp space infection
- **Route of infection:** as pulp space infection
- **Predisposing factors:** as pulp space infection
- + Thorn is derived under nail
- Trimming skin around nail

Pathology

- As pulp space infection

Clinical picture

⇒ **Symptoms:**

As pulp space infection but swelling in nail fold.

⇒ **Signs:**

As pulp space infection but in the nail fold, start one side then becomes U-shaped

Complications

- As before + spread under nail → subungual abscess

Investigations

As before

Treatment

- As before but:
 - **Incision:** oblique at the angle of nail fold
 - U-shaped if pus is present all around nail fold.
 - Nail extraction: in subungual abscess

Prognosis

- As pulp space infection

Suppurative Tenosynovitis

	Tenosynovitis in middle 3 fingers	Ulnar bursitis	Radial bursitis
Definition	Suppurative inflammation of tendon sheaths of middle 3 fingers	Suppurative inflammation of ulnar bursa	Suppurative inflammation of radial bursa
Etiology Pathology C/P	As pulp space infection		
Symptoms	But swelling all around fingers	Swelling in palms and distal parts of forearm and little finger	Swelling of thumb, thenar eminence and distal part of forearm
Signs	As pulp space infection		
	Affected finger is semiflexed (hook sign)	Slight flexion of little finger and limited movement of medial 4 fingers + edema of dorsum of the hand	Thumb is semiflexed
Complications Investigations Treatment	As pulp space infection		
	But incision is transverse over proximal cul de sac Counter incision distally in severe cases	Incision along radial border of hypothenar eminence Counter incision at lower part of forearm in severe cases	At ulnar side of thenar eminence stopped proximally 1.5 inch distal to distal wrist crease Counter incision in severe cases
Prognosis	As pulp space infection		

	Superficial midpalmar space infection	Deep mid palmar space infection
Anatomy	Anterior → palmar aponeurosis Posterior → flexor tendons	Anterior → flexor tendons Post → fascia covers interossi Lateral → fibrous band from palmar aponeurosis to 3 rd metacarpal Medial → fibrous band from palmar aponeurosis to 5 th metacarpal
Definition	Suppurative inflammation in superficial mid-palmar space	Suppurative inflammation in deep midpalmar space
Etiology	As pulp space infection	
Pathology	As pulp space infection	
Clinical picture	With obliteration of palm concavity with edema of dorsum of hand	Obliteration of palm concavity with edema at dorsum of hand with semiflexion of medial 3 fingers
Complications	As pulp space infection	
	+ formation of collar stud abscess through formation of subcuticular whitlow	+ web space infection Thenar space infection
Treatment	Incision over site of maximum tenderness	Transverse incision over the space Counter incision from the web space if affected
Prognosis	As pulp space infection	

	Web space infection	Thenar Space infection
Anatomy	- Triangle region between dorsum and ventral skin present at bases of fingers - Contain fat, Vs, Ns, and muscles	- Anterior → thenar muscles, radial bursae - Posterior → adductor pollicis - Medial → deep midpalmar space - Distal → extend to the web of the thumb
Definition	Suppurative inflammation of web space	Suppurative inflammation of web space
Etiology	As pulp space infection	
Pathology	But route may be from adjacent web spaces or deep mid palmar space	Route from tenosynovitis and deep midpalmar space
Clinical picture	As pulp space infection	
	As pulp space infection	
	Edema in web space (sign of victory)	Ballooning if thenar eminence
	As pulp space infection	
Treatment	But incision is transverse over the web one cm from edge Severe cases counter incision posteriorly	- Drained through an incision at the site of maximal tenderness or where pus points at the skin. - An incision is done along the medial side of the thenar eminence should stop 2 cm distal to the distal wrist crease to avoid injury of the motor branch of medial nerve.
Prognosis	As pulp space infection	

Ingrowing toe nail

Definition

- Necrosis of the nail sulcus due to persistent contact of the edge

Incidence

- One of the common surgical complaints usually occurs at big toe

Etiology

- Nail abnormalities as hypercurved nails.
- Bad trimming of the nail.
- Wearing tight shoes.

Pathology

- Suppurative inflammation in the nail fold with edema, redness and inflammatory cells.

Clinical picture

- Pain: in the affected toe.
- Swelling: of the ingrowing toe nail.
- Disturbance of function: limited mobility.

Investigations

- CBC, C & S.

Treatment

⇒ Conservative:

- Separate the nail from the nail bed by gauze soaked in antiseptic lotion.
- Correct trimming of the nail.
- Keep the foot dry & clean.
- Avoid tight shoes.

⇒ Surgical:

- Wege excision (but recurrence is common)
- Excision of the nail with periosteum (best results).

Prognosis

- The best prognosis with excision of the nail with periosteum.

Complications of Hernia

- Hernia is protrusion of viscous or a part of viscous usually within a peritoneal sac through a defect in abdominal wall.
- It may be congenital or acquired.
- The patient gives history of painless reducible swelling in anatomical site of hernia which recently **BECOMES COMPLICATED** by one of the following:

1- Irreducibility

- Failure of hernial contents or part of it to return to abdomen.

⇒ Causes:

1. Adhesions
2. Narrow neck.
3. Sliding hernia.
4. Over crowding of the contents.

⇒ Clinically:

- The hernia becomes **irreducible**
- **Expansile impulse** on cough

⇒ Treatment: surgical repair (but not as urgent as strangulation) with no use of truss as it may lead to strangulation

2- Strangulation

⇒ Definition: Interference with blood supply of contents with impending gangrene within 4-6 hours.

⇒ Incidence:

- Older age > infants
- Inguinal hernia (commonest) → so **the commonest** strangulated.
- Femoral hernia (narrow neck) → **so the most liable** to be strangulated.

⇒ Causes:

- Sharp edge of defect or narrow neck.
- Sudden expulsion or overcrowding of contents.
- Irreducibility, inflammation, obstruction predispose for strangulation.

⇒ Pathology:

- Venous obstruction → congestion of intestine → serous transudate → arterial obstruction → ischemia of intestine & gangrene → bloody exudate → secondary infection of fluid → finally gangrene occurs → peritonitis → septic shock.

⇒ Clinical Picture:

- History of painless reducible swelling which becomes painful (pain is colicky and stabbing) which is the cardinal symptom.
- Manifestations of intestinal obstruction
(Projectile vomiting, absolute constipation, colic, distension).
- Variable degrees of shock (hypovolaemic or neurogenic).
- The hernia becomes **irreducible**.
- **No** impulse on cough.
- Hernia is **tense & tender**.

Strangulation without obstruction may occur in:

- 1- If content is omentum.
2. Richter type.
- 2- Litter's type (Strangulated Meckle's diverticulum).

⇒ **Treatment:** *Urgent operation after pre- operative preparation.*

1. Pre- operative preparation (R & M):

1- Resuscitation (Mandatory):

- Ryle's tube → for G.I.T. suction.
- Cannula & IV fluid to correct hypovolemia.
- Pre operative medications (Morphia & Antibiotics).
- Catheter → Detection of urine output.

2- Monitoring: Pulse, temperature & blood pressure.

2. Rules of operation:

1. General anesthesia
2. Incision is wide & exploratory.
3. Expose the sac.
4. Opened at fundus (umbilical & para umbilical H → near its neck).
5. Toxic fluid content is sucked.
6. The constricting ring should be divided over grooved director or finger to avoid injury of intestine.
7. If contents viable intestine → Reduce into abdomen
8. If contents non viable intestine
 - Left side of Large intestine → resection & exteriorizations
 - Right side of large intestine or small intestine → Resection & anastomosis
9. If contents are omentum → excise it viable or not.
10. Herniotomy & herniorraphy
(*no hernioplasty → as the tissues are potentially infected*).
11. Closure in layers.

3. Post-operative management:

- a-continue ryle tube suction+IV fluids for 2-3 days.
- b-prophylactic antibiotics.
- c-remove drains when they stop discharge(5 days).

NB. TTT of predisposing factors to avoid recurrence e.g. chronic bronchitis.

3- Obstruction

- Usually in irreducible hernia.
- Lumen is obstructed by faecolith or band of adhesion.
- **No interference** with blood supply.

⇒ **Clinical Picture:** (*it is very difficult to differentiate between obstruction and strangulation*)

- Manifestations of intestinal obstruction: projectile vomiting, absolute constipation, colic.
- Variable degrees of shock
- The hernia becomes **irreducible**
- **No (or weak)** impulse on cough
- But the hernia is **not tense**

⇒ **Treatment:** Urgent operation to avoid strangulation.

4- Inflammation

- Inflammation of:
 - The wall (truss)
 - or
 - The content (e.g. appendix or Meckel's)

⇒ Clinical Picture:

- General → FAHM
- Local →
 - The hernia becomes **irreducible** & tender But **not tense**.
 - The overlying skin is red & edematous

⇒ Treatment: removal of the septic focus as the appendix.**5- Hydrocele of the Hernial Sac**

- Obliteration of the defect by adhesion or part of the omentum after reduction of the content followed by fluid collection.

⇒ Clinical Picture:

- a- **No expansile** impulse on cough
- b- **No reduction**.
- c- +ve fluctuation test.

⇒ Treatment: Excision**6- Rupture of hernial sac****7- Torsion of the omentum****8- Recurrent hernia****9- Sliding hernia**

Inguinal Hernia

Anatomical views⇒ Inguinal canal:

- It is an oblique passage in the lower abdomen formed by passage of testis from the abdomen to the scrotum.
- It descends downwards, forwards and medially from deep to superficial ring, it is 1.5 inch in length

⇒ Triangle of Halsted

It is bounded by:

- Laterally: inferior epigastric artery
- Medially: lateral border of rectus sheath
- Inferiorly: medial ½ of inguinal ligament

Definition

- Reducible swelling gives impulse on cough at inguinal region

Types

- Oblique inguinal hernia: pass through inguinal canal
- Direct inguinal hernia: through triangle of Halsted
- Dual hernia: direct and indirect types in the same side
- Sliding hernia

Incidence

- Oblique type is the commonest but direct is less common

Etiology

1- Predisposing factors:

- Congenital: as un-obliterated processus vaginalis in OIH
- Acquired: weak abdominal wall and fascia transversalis or defect in conjoint tendon

2- Precipitating factors:

- Increase in intra-abdominal pressure as in HSM, ascites or BPH

Pathology

	Oblique inguinal Hernia	Direct inguinal hernia
Defect	Internal ring lateral to inferior epigastric artery	Triangle of Halsted medial; to inferior epigastric artery
Sac	Inside the cord	Outside and behind it
Contents	Small intestine, omentum or both	
Coverings	- Internal spermatic fascia - Cremasteric muscle - External spermatic fascia - Campers, Scarpa's fascia or Detrosm if in scrotum - Skin	- Fascia transversalis - Stretched conjoint tendon - External spermatic fascia - Campers, Scarpa's fascia - Skin

Clinical picture

1. Type of patient:

- a- OIH: any age and sex
- b- DIH: elderly and males

2. Clinical picture of the hernia:

- Painless reducible inguinal swelling

	OIH	DIH
Side	Unilateral or bilateral	Usually bilateral
Site	Inguinal or inguinoscrotal	Inguinal
Size	May be large	Usually small
Shape	Oblong	Hemispherical
Descent	Downward forward and medially	forwards
Reducibility	Upwards backwards and laterally	backwards
Reaching scrotum	May occur	Very rare
Internal ring test	Hernia not protrude	Hernia protrude

3. Clinical picture of the cause: BPH, HSM or ascites

4. C/P of complications e.g. obstruction, strangulation or irreducibility.

DD

- Femoral hernia, hydrocele, varicoele

Investigations

- **Hernia is a clinical diagnosis**
- Investigations for precipitating factors and complications and preoperative e.g.
 - CXR
 - LFTS and KFTs
 - Abdominal and rectal U/S.
 - CBC, FBS and ECG

Treatment

A- Non-complicated cases:

Avoid and treatment of precipitating factors as chronic cough, BPH

1- Oblique inguinal hernia: surgical correction by

- a- herniotomy: in congenital hernia
Open at fundus, reduce content, transfix then remove sac
- b- Herniorrhaphy: done in adults and elderly patients
- c- Herniotomy + repair of posterior wall of inguinal canal (Marcy's, Bassini or Shouldice)
- d- Hernioplasty: done for
 - Very weak wall
 - large defect
 - Recurrent hernia
 Done by Darning or grafting (natural or synthetic)

2- Direct inguinal hernia

- A- repair of posterior wall and inguinal canal by Marcy or Shouldice repairs
- B- Mesh hernioplasty
- C- Truss of unfit persons for surgery

B- Complicated cases: treatment of complications

Prognosis

- Recurrence occur if PPT factors are not treated

Femoral Hernia (FH)

Anatomical view

- Femoral hernia passes through femoral canal which is the most medial compartment of femoral sheath
- It is cone shaped and about 1.5 inch
- The inlet is surrounded by sharp borders of 3 ligaments (e.g. lacunar)

Definition

- Loop of intestine or another part of abdominal contents that has been forced out of the abdomen through femoral canal.

Etiology

A- Congenital femoral hernia: (of Clouquet)

- Penetrating aponeurosis of pectineus muscle.
- Usually associated with congenital hip dislocation.

B- Acquired femoral hernia:

1. More common in females due to wide pelvis (wide femoral canal)+small blood vessels.
2. Higher incidence on the Rt. side.
3. Increase intrabdominal pr. (pregnancy, obesity, ascitis...etc).
4. Laxity of abd muscles & tendons (e.g. as in pregnancy).
5. Vasodilation & vasoconstriction of femoral Vs. → Weakness of fat.

Pathology

⇒ Coverings

1. Femoral sheath (when small).
2. Cribriform fascia (when it comes outside the saphenous opening).
3. Superficial fascia (Scarpa's fascia).
4. Subcutaneous fat.
5. Skin.

⇒ **Defect:** through femoral ring

⇒ **Contents:** omentum, bowel or both.

Clinical Picture

Type of patient:

20- 40 years old patient and usually female

Symptoms:

- 1 Symptoms of the Precipitating factors.
- 2 Pain: Painless unless complicated
- 3 Swelling: reducible swelling in the upper part of the thigh or in the groin (onset, etc).

Signs:

- 1 Swelling in upper part of the thigh or over the lat part of inguinal ligament if large hernia.
- 2 It lies below & lateral to the pubic tubercle.
- 3 Reducible downwards & medial, then backwards then upwards.
- 4 Signs of precipitating factors.

DD

Irreducible	Reducible
- Skin tumors	- Saphena Varix
- Subcutaneous lipoma	- Femoral a. aneurysm
- L.N.	- Psoas Abscess
- Mal descended testis	- Inguinal hernia
	- Psoas abscess (Cystic swelling disappear on flexion of the hip. + osteoarthritis).

Strangulated F.H.

1. Acute Lymphadenitis
2. Abscess.
3. Torsion in mal descended testis.
4. Rupture adductor longus tendon.
5. Anterior dislocation of the hip joint.

Complications

- Strangulation: more liable as the neck is narrow and surrounded by sharp borders of 3 ligaments (e.g. lacunar)
- Obstruction
- Irreducibility

Investigations

Hernia is a clinical diagnosis, preoperative investigations and investigations for precipitating factors should be done:
CBC, FBS, KFTs, LFTs, ECG, CXR.

Treatment**A. Prophylactic(Life-style measures):**

- 1 Regular daily exercise to strength abdominal muscles.
- 2 Avoid constipation
3. No smoking
4. Loosing weight if over-weight
5. Avoid lifting heavy loads.

B. Therapeutic (Surgical correction)

1. Low approach may injure the abnormal obturator artery.
2. Inguinal approach
3. Mc avedy.

Prognosis

- Depends on correction of precipitating factors and absence or presence of complications

Umbilical Hernia**Congenital Umbilical Hernia (exomphalos)****Cause**

- Failure of mid gut to return to abdomen (defect in ant. abdominal Wall).

Types

- **Exomphalos minor** → small sac & central cord, small defect (<5 cm).
- **Exomphalos major** → large sac & eccentric cord, larger defect (>5 cm).

Covering

- Amniotic membrane, Wharton's jelly.

Treatment

1. Operation within 1st hrs of life: (Undermine skin; Close it, ± 2 release incisions).
2. Then repair of hernia might be delayed for months or even years
3. Non operative TTT: (poor general condition) → alcoholic mercurochrome 2%.

Infantile umbilical hernia**Cause**

- It occurs few wks or ms after birth (weak umbilical scar).

Covering

- Stretched umbilical scar.

Treatment

- Treatment of the PPT factors.
- Coin & plaster strap.
- Surgical correction (large defect, > 4 yr, strangulation)

Para Umbilical Hernia**Cause**

- Defect in linea alba just above (commoner) or below (less common) umbilicus,

Covering

- Skin, subcutaneous tissue, extra peritoneal fat.

Clinical Picture

- See before +
- Local examination:
 - i) Swelling above or below umbilicus (sac is multiloculated).
 - ii) Partially irreducible or reducible directly backwards.
 - iii) Intertrigo affecting overlying skin.

Treatment

- **Treatment of the precipitating factors**
- **Surgical correction** (Anatomical repair, Mayo's repair).

Epigastric hernia

- Occurs in linea alba but separated from umbilicus by interval.

Clinical Picture

- **Asymptomatic**, Impulse on cough is usually absent early.

Treatment

- Treatment of the precipitating factors.
- Surgical repair (Mayo's repair, Anatomical repair).

Recurrent hernia

- After repair of any type of hernia, (10 - 15%)

Causes

Pre- operative	Operative	Post operative
i) Weak abdominal ms.	i) Excessive trauma to tissue.	i) Wound infection.
ii) Obesity.	ii) Bad hemostasis.	ii) Vomiting.
iii) Ch. cough	iii) Incomplete removal of sac.	iii) Early return to work.
iv) Ch. constipation	iv) Missed sac	iv) Persistent ppt. factors
v) SEP.	v) Repair is too loose or too tight.	
vi) Anemia	vi) Using absorbable threads.	

Incisional Hernia

- Immediately or early in the post operative I (man made hernia).

Causes

Pre- operative	Operative	Post operative
i) Weak abdominal ms.	i) Excessive trauma to tissue.	i) Wound infection.
ii) Obesity.	ii) Bad hemostasis.	ii) Vomiting.
iii) Ch. cough	iii) Repair is too loose or too tight.	iii) Early return to work.
iv) Ch. constipation		iv) Persistent ppt. factors
v) SEP.		

Treatment

- **Prophylactic treatment:**
 - i) Avoid PPT factors.
 - ii) Use of non absorbable prolene sutures in closure of abdominal incision.

- **Surgical correction:**

1- Anatomical repair. 2- Maingot (Keel) repair.

- **Abdominal corset:** if operation is contraindicated.

Dual hernia (pantaloon) (saddle bag)

- Both direct & indirect inguinal hernia arises in the same side with the inferior epigastric vessel between the neck of the 2 sacs.

Sliding Inguinal Hernia (Hernia en glissade)

- In patient with long standing hernia.
- The peritoneum of the viscous forms a part of the sac.
- Caecum, pelvic colon, bladder might slide.
- Diagnosis usually intra operative.

Burst abdomen

Definition

- Complete disruption of an abdominal incision in the early post-operative period.

Etiology

Etiology of incisional & recurrent hernia is the same as burst abdomen

Types

- **Partial:** deep layers burst but the skin is intact producing → incisional hernia.
- **Complete:**
 - a) If the intestine prolapsed out of the wound → called evisceration.
 - b) If the intestine is retained inside abdomen → called wound dehiscence.

Clinical picture

- Usually occurs at 6th -8th day postoperative.
- A serosanguinous discharge is often a warning sign and called → **Red sign** (most important)
- The patient feels as if something gives away.
- Symptoms of intestinal obstruction may be present.

Treatment

- **Prophylaxis:** Avoid and treat any predisposing factor.

- **Active TTT**

⇒ **Complete burst**

- **Pre-operative measures:**
 1. Cover the wound with a sterile towel and warm saline.
 2. Ryle's tube + IV fluids + antibiotics.
- **Operative:**
 1. Protruded intestinal loop are washed with saline and returned to the abdomen, the omentum is spread over the intestine, the abdominal wall is closed as one layer using **tension "through and through" suture**.
 2. Abdominal binder is used post-operative.

Divarication of recti abdominis

- Elderly multipara → when pt. Strains → a gap between recti through which abdominal contents bulge.
- Relaxed abdomen → fingers can be introduced between recti.
- **Treatment:** an abdominal belt is all that is required.

Maydl's hernia (W- hernia)

- The gangrenous loop is found in the abdomen as there are double loops in the hernial sac.

Richter's strangulated hernia

- Gangrene without obstruction → gangrene of the antimesenteric border of the intestine.

Rare types of hernia

Spigelian hernia

- Old ♀, defect in semilunar line at level of arcuate line → contains fat later on full peritoneal sac.
- Liable to strangulation → anatomical repair (treatment of choice).

Lumbar hernia

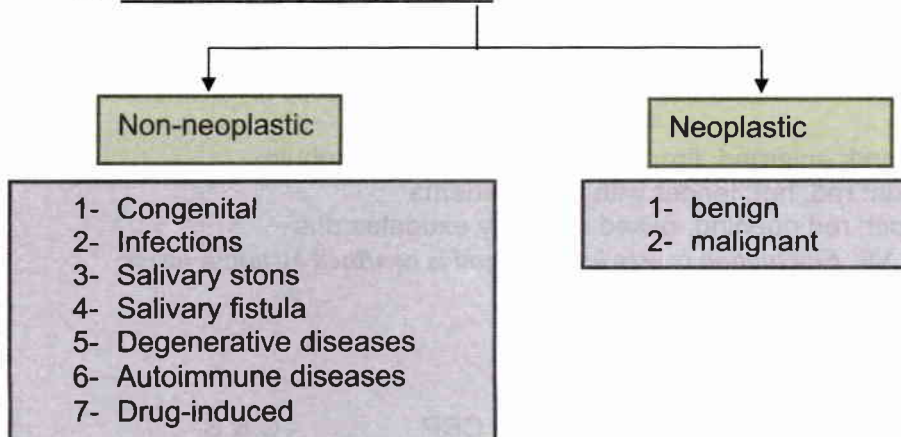
- In superior or inferior lumbar triangle.
- Large & reducible, with impulse on cough & has a wide neck.
- Treatment by abdominal corset or surgery.

Perineal hernia

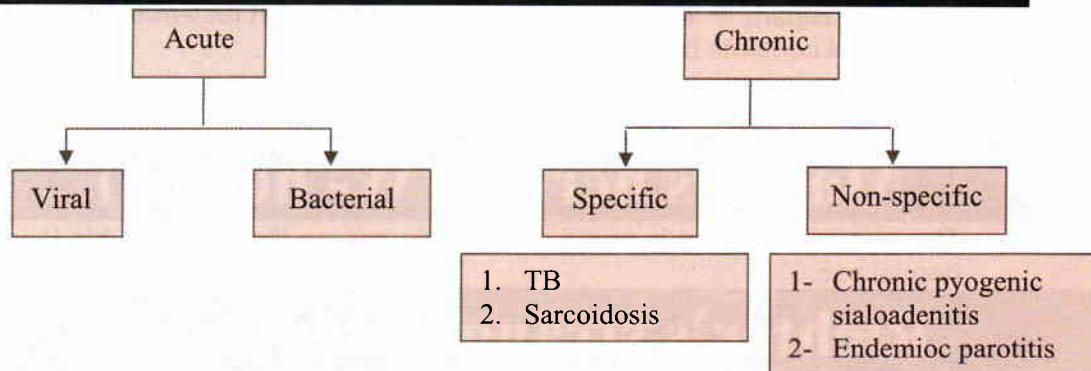
- After abdominoperineal operation.
- It occurs at the surgically obliterated site of the previous anus.

Salivary Glands

⇒ Disease of salivary Glands



Infections



Acute bacterial Sialadenitis

Anatomical view

- There are 3 pairs of main salivary glands (parotid, submandibular and sublingual) together with other glands scattered in mucous membrane of the mouth.

Definition

- Acute suppurative inflammation of the salivary glands (e.g. parotid)

Etiology

- Causative organism:** mainly staph aureus
- Predisposing factors:** poor hygiene and postoperative
- Precipitating factors:** duct obstruction
- Route:** direct (duct) or blood

Clinical picture

⇒ Symptoms

General: FAHM

Local:

- Pain in the side of the face.
- Swelling on the affected side
- Disturbance of function as pain ↑ with ingestion of lemon.

⇒ Signs

General:

- Toxemia may be marked after suppuration, hectic fever and tachycardia

Local:

- Gland: enlarged, firm, tender and raising ear lobule
- Skin: red, hot, tender with pitting edema
- Duct: red opening, raised and may exudates pus

NB. Fluctuation is very late as gland is confined by tough fascia

Complications

- Chronicity, abscess, stone, fistula

Investigations

- CBC → leucocytosis
- ESR ↑
- ↑ CRP
- C & S

Treatment

A- Prophylactic: avoid predisposing factors

B- Curative:

- Early: hot fomentation +3A (antibiotic, analgesics, antipyretic)
<The best antibiotic is clindamycin d.t high concentration in the saliva>
- Late(failure of response to antibiotic after 48hrs or evidence of suppuration): surgical drainage
→ In parotid abscess: Blair's incision & Hilton's technique

Postoperative complications: include fistula formation. Facial nerve injury & frey's postgustatory syndrome

Salivary Stone

Incidence

- Submandibular : parotid = 50:1 d.t.:
- Submandibular gland secretions are more viscid.
- Submandibular salivary gland is in a dependant position and duct may be blocked by food particles.

Predisposing factors

- Chronic infection and stasis (duct obstruction)

Pathology

- Stones are single or multiple and may be in the gland or the duct

Clinical picture

- Pain: salivary colic especially during meals may referred to tooth, ear or the tongue
- Swelling: of the affected gland

Complications

- Sialectasis, Sialadenitis or fistula.

Investigations

- Plain X-ray and sialography

Treatment

- Stone in the duct → duct is opened and stone is extracted
- Stone in the gland:
 - Submandibular → submandibular sialadeectomy
 - Parotid → superficial conservative sialadeectomy

Salivary Neoplasm

Pleomorphic Adenoma (Mixed Tumor)

Incidence

- Commonest in the parotid, less in submandibular gland.

Pathology

- Benign tumor.

Site

- Usually from superficial part of parotid gland.

Macroscopic

- Firm, multicentric(incomplete capsule), grayish white.

Microscopic

- **Cells:** epithelial cells arranged in sheets or duct-like structures.
- **Matrix:** blue-stained mucinous material (pseudo-cartilagenous).

Clinical features

Symptoms

- Painless, slowly growing swelling in the side of the face.

Signs

- Swelling characterized by being:
 - Well-defined, lobulated, freely mobile (not attached to skin, muscles or bones)
 - variable in consistency (firm or cystic but never hard) elevating lobule of ear
 - with no cervical LNs enlargement or fascial nerve infiltration

DD

- Non-neoplastic lesions, LNs, sebaceous cyst and lipoma.

Complications

- 1- Disfigurement.
- 2- Malignant transformation after > 10 years is rare (2-3%).

Investigations

- 1- CT scan for assessment of tumors arising from deep part of the parotid.
- 2- Tc99 scan: cold spot.

Treatment

1- Tumor in parotid:

- a- Conservative superficial parotidectomy: if the lesion is superficial.
- b- Total conservative parotectomy: if the lesion is deep.

2- Tumor in submandibular gland:

submandibular sialadenectomy.

N.B: Enucleation of the tumor is followed by a high recurrence rate.

Monomorphic Adenoma (Adenolymphoma, Warthin's tumor)

Incidence

- 10% of parotid tumors and bilateral in 10% of cases especially in old age and smokers

Pathology

Site

- Superficial part and lower lobe of parotid gland

Macroscopic

- Cystic, encapsulated benign tumor

Microscopic

- Epithelial lines spaces filled with creamy material and surrounded by lymphoid tissue

Clinical picture

Symptoms

- Elderly male > 50 years, presents with painless, slowly growing swelling in side of the face.

Signs

- Same as pleomorphic adenoma but always cystic or soft in consistency, in front of tragus of ear, not elevating lobule of ear

DD

- See pleomorphic adenoma.

Investigations

- 1- Tc99 scan: hot spot
- 2- CT scan

Treatment

- Conservative superficial parotidectomy or better enucleation of the tumour.

Carcinoma of salivary glands

Etiology

- 1- De-novo.
- 2- On top of mixed parotid tumor.

Pathology

Macroscopic

- Non-encapsulated infiltrating tumor with grayish areas of hemorrhage and necrosis

Microscopic

a. Mucopidermoid carcinoma:

- The commonest type, arises from the duct epithelium, consists of sheets of columnar (mucoid) and squamous (epidermoid) cells of 3 grades of differentiation (low, intermediate, high)

b. Adenoid cystic carcinoma (Cylindroma)

- (Swiss-cheese pattern) → composed of myoepithelial cells (basophilic) + epithelial cells (eosinophilic).

c. **Acinic cell carcinoma**

- Composed of serous acini (so occur only in parotid gland)

d. **Adenocarcinoma:**

- Rare, 3 basic histological patterns (tubular, papillary and undifferentiated)

e. **Carcinoma ex pleomorphic adenoma**f. **Undifferentiated****Spread**

- 1- **Direct:** to the surroundings.
- 2- **Lymphatic:** to upper deep cervical LNs.
- 3- **Blood:** rare & late.

Complications

- | | |
|----------------|-----------------|
| 1- Ulceration. | 2- Infection. |
| 3- Hemorrhage. | 4- Nerve palsy. |

Clinical Picture**Symptoms**

- 1- **Pain:** may radiate to the ear, increase by mastication.
- 2- **Swelling:** rapidly growing on the side of the face.
- 3- **Disturbance of function = complications.**

Signs

- 1- **Swelling:**
 - Usually warm and mildly tender.
 - Ill-defined edge.
 - Rapid rate of growth.
 - Infiltration of skin, muscles, vessels & nerves.
- 2- **Cervical LNs:** enlarged, stony hard, mobile then fixed.

Investigations**For diagnosis**

- | | |
|--------------------------|----------------------------|
| 1- Tc99 scan → cold spot | 2- Biopsy → from the gland |
|--------------------------|----------------------------|

For staging

- | | |
|---|------------------|
| 1- CT scan to determine local extent of tumor | 2- FNAB from LNs |
| 3- Metastatic work up | |

Treatment**Operable**

⇒ **According to site of tumor:**

- 1- **Cancer in parotid gland:**
 - Total radical parotidectomy + total block dissection of the neck LNs + postoperative radiotherapy to decrease recurrence
- 2- **Carcinoma in submandibular gland:**
 - Commando operation

Inoperable

- Palliative resection + radiotherapy.

Swellings

Lipoma

Definition

- It is a benign tumor arising from adipose tissue.

Pathology

Site

⇒ The lipoma is classified according to site into:

1. Subcutaneous type:
 - ◆ The commonest type.
 - ◆ Common in back, shoulder, buttocks, forehead and limbs.
2. Subfascial type:
3. Submucous lipoma: Beneath mucous membrane of larynx, intestine or stomach.
4. Subserous lipoma: Beneath visceral or parietal peritoneum or pleura.
5. Subsynovial lipoma: Beneath synovial membrane of the joint.
6. Inter and intramuscular lipoma: In between or within the muscle.
7. Subperiosteal lipoma: Deep to periosteum of flat bones or long bones.
8. Intraglandular lipoma: Inside the gland as parotid gland, thyroid gland and breast.
9. Extradural lipoma:
 - ◆ Arises in relation to spinal cord.
 - ◆ Never in the cranium: as there is no fat.
10. Retroperitoneal lipoma: Behind post parietal peritoneum.

Pathological classification

- Lipoma is classified according to structure into:
 - a. Pure lipoma: mainly adipose tissue.
 - b. Fibrolipoma: with extensive fibrous tissue.
 - c. Hemangiolipoma or navio lipoma: with extensive vascular tissue.
 - d. Myxolipoma.

Gross picture

- Size: variable.
- Shape: Oval or rounded.
- Surface: Lobulated.
- Consistency:
 - ◆ Soft.
 - ◆ Firm in Fibrolipoma.
- Color: Yellowish.
- Cutsection: thin fibrous capsule sends fibrous tissue septa divide the tumor into multiple spaces.
- Capsule: 2 capsules:
 - ◆ Inner true: Formed of fibrous tissue.
 - ◆ Outer false: Formed by compressed surrounding structures.
 - ◆ Between the 2 capsules there is a line of cleavage that facilitates enucleation of the tumor.

- Origin: adipose tissue.
- Blood supply: through the pedicle at one side of the tumor.
- Number:
Usually single but may be multiple e.g. Dercum's disease

Microscopic picture

- Aggregation of fat cells with signet ring appearance. Separated by fibrous connective tissue stroma containing blood vessels arising from the pedicle.

Clinical picture

- It differs according to the site.

Complaint

- Painless slowly growing swelling (but may be painful in neurolipoma or complicated lipoma)
- Cosmetic disfigurements.

Signs

- General: e.g. fever if lipoma is complicated by infection.
- Local: According to site:

1. Subcutaneous lipoma(the commonest):

◆ Inspection:

- ▲ Rounded or oval swelling may attain large size, common at shoulder, back, buttocks with normal skin overlying.

◆ Palpation:

- ▲ No tenderness, no hotness with lobular surface and well defined slippery edge (diagnostic) "It moves in front of my advancing finger".
- ▲ Soft & may give pseudofluctuation.
- ▲ Attached to skin: dimpling on pinching or gliding the skin over it.
- ▲ Lymph nodes are not affected.

2. Subfascial lipoma

◆ Inspection:

- ▲ Rounded or oval swelling, common at forehead with normal skin overlying.

◆ Palpation: as subcutaneous type but:

- ▲ Lobulation can not be felt.
- ▲ Not attached to overlying skin.
- ▲ Firm in consistency, no slippery edge.

3. Sub synovial lipoma

◆ Inspection:

- ▲ Rounded or oval swelling related to a joint with normal skin overlying.

◆ Palpation:

- ▲ As subcutaneous type but not attached to skin.

4. Submucous lipoma

- ◆ Beneath mucous membrane of larynx → it may cause respiratory obstruction.
- ◆ Beneath mucous membrane of the intestine → it may cause intussusceptions.

5. Inter or intramuscular

- ◆ Swelling in or in between muscles, common in thigh, shoulder with normal skin overlying.
- ◆ It becomes more firm on muscle contraction.

6. Subperiosteal lipoma

- ◆ Swelling in relation to long or flat bones.
- ◆ It is difficult to be differentiated from osteoma.

7. Extradural:

- ◆ Only in spinal cord(never in cranium as there is no fat).
- ◆ Presented by pressure symptoms.

8. Intraglandular: inside agland as parotid,breast or thyroid.**9. Subserous:** beneath visceral or parietal peritoneum or pleura**10. Retroperitoneal:**

- Behind post. Parietal peritoneum
- It may turn malignant.

Complications

1. Infection.
2. Ulceration of overlying skin and mucous membranes.
3. Degenerative changes, liquefaction or calcification.
4. Pressure symptoms e.g. extradural lipoma.
5. Malignant transformation: rare except in retroperitoneal lipoma.

Investigations

- Specific:
 - Excisional biopsy.
 - X-Ray: if Subperiosteal.
 - CT spinal cord if it is extradural.

DD

- According to its site e.g. Subperiosteal should be differentiated from osteoma.

Treatment

- Surgical excision(enucleation) if:
 - Not accepted cosmetically.
 - Complicated.

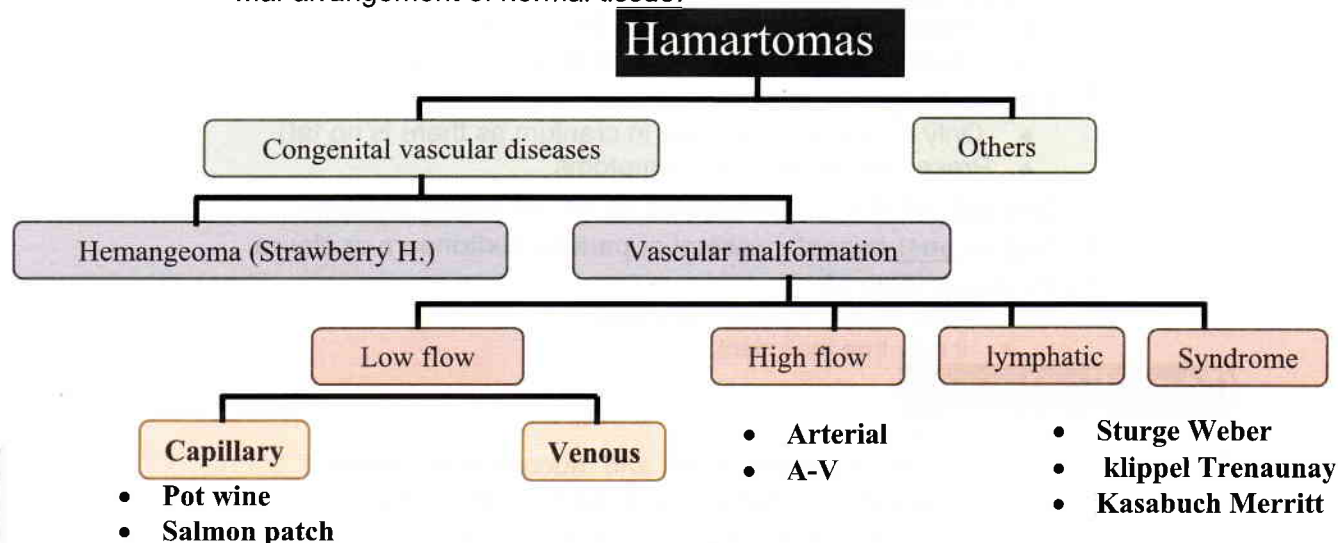
Prognosis

- Subcutaneous lipoma never to turn malignant, but in thighs and buttocks there is increased incidence of malignant transformation.
- Retroperitoneal lipoma: more variable to turn malignant.

Hamartoma

Definition

- Mal-arrangement of normal tissue.



Congenital Vascular Diseases

A-Hemangioma (strawberry hemangioma)

Definition

- Benign tumor of endothelial cells of capillaries.

Incidence

- Male : Female = 3 : 1

Pathology

- Site: Anybody surface but the most common site is face.
- The lesion usually presents by the following 3 stages:
 - Stage of proliferation.
 - Involution phase.
 - Involved phase.

Clinical picture

- Lesion start as erythramtus raised patch with irregular surface
- During the 1st 2-3 wks after birth.
- Then it grows Stationary for 2-3 yrs.
- Finally spontaneous regression starts & completed by the age of 5-7 yrs.

Complication

1. In the face → Squint.
2. In the eye lid → ↓ visual field → amplyopia + blindness.
3. Ulceration is a common compliction.
4. Bleeding is not common.
5. Infection .
6. Air way obstruction.

Treatment

1. Reassurance of the parents and conservation: waiting for spontaneous regression.
2. Surgical interference:
 - ⇒ Indications:
 - a. lesion affecting the visual field (to avoid squint or diplopia).
 - b. rapidly growing lesions.
 - c. ulcerated lesions.
 - ⇒ Technique:
 - a. Intralesional corticosteroids.
 - b. Laser photo coagulation.
 - c. Surgical excision (followed by reconstructive surgery).
 - ⇒ Disadvantage: scar of excision (compared by minimal scar of involution).

B. Vascular malformations**I- Low flow Malformations****A. Capillary malformations****I- Portwine stain****Incidence**

- Commonest type of vascular malformation

Clinical picture

- The lesion is present since birth (deep purple in colour & not raised) and it doesn't undergo involution.
- It may take the distribution of one of the branches of trigeminal nerve, but the lesion doesn't cross midline.
- Sometimes a portwine stain of the face is associated with similar lesions in the meninges (Sturge Weber Syndrome).

Treatment

- I. Conservative: main line
- II. Surgical:
 - a. Embolization.
 - b. Intralesional corticosteroids or sclerosing agent.
 - c. Laser photocoagulation.
 - d. Surgical excision.

II- Salmon Patch

- Nearly half of all babies have salmon patch.
- Appear as pink, flat marks on the forehead, back of the neck or on the lip.

B. Venous Malformations (Previously called cavernous hemangioma)

Definition

- Multiple large inter communicating sinus (vascular spaces)

Incidence

- less common than capillary (portwine)

Site

- External: Cheeks, lips Tongue, eye, ear extremitiesect
- Internal: the commonest site is liver.

Clinical picture

- 1- Present since birth / no spontaneous involution.
- 2- Compressible swelling with some discoloration of overlying skin.
- 3- Sign of emptying is very characteristic: Pressure causes blenching but colour returns immediately after release of pressure.
- 4- If in the liver→sequestration of platelets→thrombocytopenic purpura.

Investigation

- 1- Arterioigraphy: preoperative : shows extent of the lesion (both diagnostic & therapeutic).
- 2- CT Scan : to show extent of the lesion

Treatment

- 1- Conservative:Compression to relieve pain and edema.
- 2- Surgical:
 - a.Percutaneous sclerosis with hypertonic saline or 100% alcohol,but with high recurrence rate.
 - b.Embolization:preoperative to↓ bleeding or therapeutic.
 - c.Interstitial laser coagulation.
 - d. Surgical excision:most definitive tt.

II- High flow malformations

I- Arterial (plexiform hemangioma) (Cersoid aneurysm)

Definition

- It is congenital A-V fistula.

Pathology

Site

- Usually in the scalp, especially in temporal region or occipital region and may involve underlying bone

Macroscopic picture

- A bluish cystic swelling

Microscopic picture

- Consists of arteries and veins

Diagnosis

- ♦ General:
 - Fever if inflamed
 - Water hammer pulse.
- ♦ Local:
 - **Inspection:**
 - Irregular swelling at temporal region, with normal skin overlying.
 - **Palpation:** pulsating compressible swelling.
 - **Auscultation:** machinery murmur over it.

Complications

1. Hemorrhage. very dangerous as it's arterial
2. Cosmetic disfigurement.
3. Infection.
4. Ulceration of skin overlying.
5. Degenerative changes e.g. calcification.

Investigations

- Doppler and duplex.

Treatment

- **Surgical excision with the following precautions:**
 1. Semisitting position.
 2. General hypotensive anesthesia.
 3. Preoperative embolization.
 4. Temporary ligation of the external carotid artery.

II- Arterial venous malformation

- Characterized by abnormal connections between arteries and veins with out an intervening capillary bed.
- Present at birth but may not become evident until late childhood.
- It may be localized or diffuse.
- Localized AVM presents by a localized swelling with increase surface temperature. There may be palpable thrill or murmur.
- Generalized AVM growth disturbance or skeletal distortion.

Treatment

- a. Ligation or embolization feeding vessel often results in rapid enlargement of collateral vessels increasing the size of the lesion.
- b. Excision.

NB: Hereditary haemorrhagic telengactesia is an autosomal dominant disease characterized by multiple cutaneous , visceral an mucosal AVM

III. lymphatic malformation

- Classified as microcystic or macrocystic and may have a component of venous malformation.
- The head & neck or the axilla are affected in majority of cases.
- Neck masses may extend into the mediastinum.
- LMs are the most common cause of congenital macroglossia, macrocheilia & macrootia.
- Skeletal involvement may cause distortion

Treatment

- a. Percutaneous aspiration followed by sclerosis may provide short term improvement; usually requires additional ttt.
- b. Surgical excision often requires multiple staged procedures. Usually wait until the age of 3 years.

Sebaceous Cyst

Definition

- Retention cyst of sebaceous gland.

Etiology

- Acquired cyst caused by:
 - Obstruction of sebaceous gland duct by inspissated sebum-Drills- scarring.
 - Result in: Retention of sebaceous secretion into sebaceous gland.

Pathology

Sites

- Anywhere in the skin related to hair. *Never in palms and sole "No sebaceous glands"*

Macroscopic

- Cystic swelling attached to skin at a point "Punctum"
- Contains: semisolid, greasy sebaceous, material with bad odour.

Microscopic

- A true cyst: lined by flat squamous epithelium.

Diagnosis

- Clinical picture:
 - Age: (rarely before adolescence)
 - Complaint: a slowly growing painless subcutaneous swelling.
 - Signs:
 - Inspection:
 - Site and number: hairy skin, single or multiple
 - Shape and size: hemispherical, small
 - Skin: characteristic punctum "black spot"
 - Palpation:
 - Smooth, well-defined, tense cystic "Paget's test"
 - Attached to skin at a point called punctum which is black spot
 - On squeezing discharge sebum.
 - Not compressible, not tender, not translucent

NB. If inflamed → ↑size, tender, fixed to deep structures

Complications As any cyst

- 1- Commonest → infection and suppuration
- 2- Sebaceous horn → dried inspissated sebum protrude from the punctum.
- 3- Cock's peculiar tumor (very rare) → (ulceration of sebaceous horn)
- 4- Local alopecia: due to stretch of the skin for long time.

NB. Simulate squamous cell carcinoma but base is not indurated

DD

- 1- Dermoid cyst
- 2- Cock's peculiar tumor from:
 - Squamous cell carcinoma.
 - Basal cell carcinoma

Investigations

- Not important. It is a clinical diagnosis.
- May be needed with complications:
 - 1- Biopsy: in cock's peculiar tumor.
 - 2- Culture and sensitivity with recurrent infection.
 - 3- Fasting blood sugar with recurrence of infection inspite of adequate treatment

Treatment

- 1- Uncomplicated → excision through and elliptical incision to include the punctum.
- 2- Infected → Incision and drainage
 - After inflammation subsides → excision

Dermoid Cyst

Types

- 1- Sequestration dermoid cyst.
- 2- Implantation dermoid cyst.
 - Epidermoid cyst.
- 3- Tubulodermoid cyst
 - Thyroglossal cyst.
 - Branchial cyst.
- 4- Teratomatous dermoid cyst of the ovary and testis.
- 5- Inclusion dermoid.

Sequestration Dermoid Cyst

Definition

- A congenital true cyst lined by squamous epithelium.

Etiology

- A congenital cyst.
- Caused by sequestered piece of epithelium in the subcutaneous tissue at a line of fusion of the body during fetal life but it appears later in life.

Pathology

Site

- **Along the lines of fusion:**
 - 1- Face → external angular (commonest) → internal angular
 - 2- Ear → post-auricular → pre-auricular
 - 3- Neck → in midline → sublingual
→ inframyelohyoid. → spurasternal
 - 4- Trunk → in midline

Macroscopic

- Cystic swelling.
- Contains: yellowish greasy sebaceous material.

Never in a limb (develop from buds → no line of fusion)

Microscopic

- True cysts lined by stratified squamous epithelium.

Diagnosis

Clinical picture

- Age: at childhood
- Complaint:
 - A slowly growing painless subcutaneous swelling related to the previously mentioned sites.
- Signs:
 - Inspection:
 - Site and number: see before + usually single
 - Size and shape: hemispherical + variable size.
 - Skin: normal
 - Palpation:
 - Smooth, well-defined, lax cystic "Paget's test".
 - Not tender, not compressible, not translucent
 - Not attached to skin(to differentiate it from sebaceous cyst).

Complications

As any cyst (Infection, rupture, hemorrhage, pressure)

- 1- Cerebral compression (very rare) → Dumble shaped or hourglass dermoid
- 2- Intracranial connection with the dura matter should be investigated by skull x-ray before excision to be sure the sutures are closed.
- 3- Recurrence after excision → if incompletely excised

DD

From sebaceous cyst

Investigations

- Skull x-ray → to exclude presence of bone defects (if present we wait till it closes)

Treatment

- 1- Uncomplicated → excision (best line of treatment)
- 2- Infected → incision and drainage followed by excision after inflammation subsides.

Epidermoid Cyst (Implantation Dermoid Cyst)**Definition**

- Acquired dermoid cyst

Etiology

- Implantation of skin into subcutaneous tissue by:
 - 1- Pricking wound on the tip of a finger as during sewing.
 - 2- Use of skin as graft in hernioplasty.

Pathology**Site**

- In a tip of finger.
- Related to scar traumatic or surgical.

Macroscopic & microscopic

- See before

Diagnosis

Type of patient: sewer or manual worker with a swelling related to tip of finger

Complaint: slowly growing swelling at 1st painless then painful

Signs: see before + tender due to compression on nerve endings at pulps of fingers

Complications and treatment

- See before

Inclusion demoids

- These are due to inclusion of the epidermis during closure of a cavity. They include sublingual, suprasternal, intracranial and intraspinal dermoids

Teratomatous dermoids

- These are benign forms of teratomas.
- The cyst is lined by squamous epithelium but contains teeth, hair, bone or cartilage.
- They occur most commonly in the ovary but may be found in the testis or posterior mediastinum.

Branchial Cyst

Embryology

- Normally the 2nd branchial arch grows rapidly covering the 3rd and 4th arches then fuse with the 5th arch
- Resulting in a space called (cervical sinus).

Etiology

- Cervical sinus persists → branchial cyst.
- + 2nd arch does not completely fuses with the 5th arch → congenital branchial fistula.

Pathology

Site

- Lat. Side of upper part of the neck.
- Related to carotid triangle (passes through carotid bifurcation).
- Partially deep to sternomastoid muscle protruding beneath its anterior border.

Macroscopic

- A cyst with a fibrous cord passing between external and internal carotids Connect cyst to pharynx "end above tonsil" → fossa of rosenmuller.

Microscopic

- True cyst lined by squamous epithelium.
- Contain mucoid substance rich in cholesterol (rectangular with one missing angle)
- Rich in lymphoid tissue → liable for infection.

Diagnosis

Clinical Picture

- **Age:** although congenital, it appears at the age of 20 years
- **Complaint:**
 - a slowly growing painless swelling at lateral side of the upper part of the neck
- **Signs**
 - a- **Inspection:**
 - Site and number: see before + unilateral or bilateral
 - Shape and size: oval + moderate size
 - Skin: normal or show fistula at the lower 1/3 of the anterior border of the sternomastoid muscle + crescentic shape discharging mucous or pus.
 - b- **Palpation:**
 - Smooth, well-defined, tense cystic.
 - Not tender, compressible, pulsated & not translucent.
 - Not attached to skin.
 - Moves from side to side
 - On contraction of the sternomastoid muscle it bulges out.
 - Fistulous track may be felt as a thread passing up deep to the anterior border of the sternomastoid muscle.
 - No signs of inflammation except: if infected

Complications

► As any cyst

- 1- Infection: is the commonest
- 2- Acquired branchial fistula due to rupture, incomplete excision or incomplete incision of the abscess,
- 3- Adenocarcinoma: very rare (MCQ)

DD

Swelling at lateral side of the neck

- | | |
|----------------------------|------------------------------|
| 1- Cold abscess (TB C/P) | 3- Laryngocele, pneumatocele |
| 2- Cyst in a thyroid lobe. | 4- Carotid artery aneurysm. |

Investigations

- U/S:
 - Ensure the diagnosis.
 - Exclude DD as cold abscess.
- Discharge → Zheil Nielsen stain
 - Characteristic rectangular cholesterol crystals with missing angles.
- If fistula: fistulogram.
- Routine preoperative.

Treatment

- Cyst: complete excision through transverse neck incision.
- Fistula: excision through Step-ladder operation.

Cystic Hygroma (Cavernous Lymphangioma)

Definition

- Dilated cavernous lymph spaces filled with lymph.

Etiology

- It is a hamartoma rather than true tumour of lymphatic vessels.

Pathology

- Consisted of multiple intercommunicated lymph spaces, commonest in the neck and axilla, less common in lip (macrocheilia) and tongue (macroglouassa)

Clinical Picture

- Lax cystic swelling partially compressible, non-pulstaing mass.
- Translucent (the only translucent neck swelling), large, irregular and ill-defined
- Superficial to sternomastiod & extends to posterior triangle (to DD from branchial cyst)

Complication

1. Obstructed labour.
2. Recurrent infection.
3. Respiratory distress due to compression of trachea to induce fibrosis.

Treatment

- Leave if for 2 years for spontaneous regression, then
- Surgical excision as much as possible after injection of boiling water, saline.

Ranula

- It is a retention cyst arising from sublingual salivary gland.
- C/P:
 - Cystic, bluish, and translucent swelling with prominent blood vessels over it and stretch submandibular duct.
 - Overlying: covered by m.m. & crossed by Wharton's duct.
 - May extend down in the neck → plunging ranula.
- TTT:
 - Marsupialization (deroofing) & suture cyst wall to oral mucous membrane
 - Excision (difficult) in recurrent cases.

Surgical Nutrition

Malnutrition in surgical patient

Incidence

- About 30% of patients with GIT diseases.
- It reaches 60% in those with prolonged hospital stay.

Etiology

A. Starvation (↓ input):

1. Social causes as poverty & neglected elderly
2. Loss of appetite by chronic diseases.
3. Dysphagia.
4. Repeated vomiting.
5. Malabsorption syndrome → in {
 - Chronic pancreatitis
 - Short gut syndrome
 - Chron's & ulcerative colitis
6. Post-operative restriction of oral intake (e. g. paralytic ileus).

B. Hypercatabolism (↑ output):

1. Major trauma and burns.
2. Major surgical operations.
3. Severe acute pancreatitis.
4. Major sepsis e. g. septicemia..

Diagnosis

A. Anthropometric measures.

B. Laboratory:

1. Serum albumin < 3.5 gm/L.
2. Determine nitrogen balance (whether +ve / -ve).
 - Input – output → +ve nitrogen balance (anabolism).
 - -ve nitrogen balance (catabolism).
3. TLC < $1.200 \times 10^9/L$.
4. Impaired hypersensitivity reaction.

Types of Surgical nutrition

- A. Enteral nutrition.
- B. Parenteral nutrition.

A. Enteral Nutrition

Definition

- Delivery of nutrients into the GIT.

Indications

- Patient in whom oral intake is inadequate with functional accessible GIT (as comatosed patient, severe dysphagia, neck surgery and burns).

Routes of administration

- 1- **Sip feeding:** whole food by mouth (fluid formula).
- 2- **Tube feeding techniques:**
 - i. **Nasogastric tube (NGT):** Ryle's tube
 - ii. **Gastrostomy:** if nasogastric feeding is not possible e. g. upper GIT obstruction.
 - iii. **Jejunostomy:** if the stomach is diseased.

Administration formulae

- A- Through gastrostomy → liquid diet, juice and milk.
- B- Through jejunostomy → either partially digested or elemental formulae.

Complications

- 1- **Mechanical:**
 - Malposition, displacement, blockage, breakage or leakage of the tube.
 - Erosion of skin or mucosa.
- 2- **Infective:** exogenous (handling contamination) or endogenous.
- 3- **GIT:** diarrhea, bloating, nausea, vomiting, abdominal cramps, aspiration, constipation.
- 4- **Metabolic/chemical:** electrolyte imbalance, malnutrition and drug interactions.

B. Total Parenteral Nutrition (TPN)

Definition

- Provision of all nutritional requirements through intravenous route without use of GIT.

Indications

- Seriously ill patients with malnutrition where the use of GIT for feeding is not possible as follow:
 - 1- GIT is **blocked** e.g. stricture, neoplasm or extrinsic mass.
 - 2- GIT is **short** e.g. short gut syndrome.
 - 3- GIT is **fistulated** e.g. enterocutaneous fistula.
 - 4- GIT is **inflamed** e.g. inflammatory bowel disease.
 - 5- GIT can **not cope** e.g. severe trauma or hyper-catabolic state.

Route of Administration

- **Central:** catheter inserted via subclavian or internal or external jugular vein.
- **Peripheral:** (appropriate for short term feeding up to 2 weeks)
 - a-Peripherally inserted central venous catheter (PICC) line.
 - b-Conventional short cannula in the wrist vein (isotonic fluid).

Complications

- Nutritional and metabolic complications resulting in overfeeding, underfeeding or electrolyte imbalance .
- Catheter complications:
 1. Misplacement of the catheter.
 2. Insertion of a catheter tip in the pleural cavity leading to haemothorax or pneumothorax.
 3. Injury to arteries or to nerves of brachial plexus.
 4. Venous thrombosis.
 5. Central venous catheter infection & septic thrombophlebitis which lead to septicemia and if neglected death..

Monitoring of the patient

- **Daily:** → urine output, Blood (Urea, Electrolyte, Glucose).
- **Twice weekly:** → Ca, Ph, Albumin, CBC
- **Weekly:** → LFT, Plasma lipids, Magnesium.
- **Monthly:** → foliate, Iron, Zinc, PT

2

THYROID



Investigations

I- For diagnosis:

A- laboratory (thyroid function tests):

- 1- Total t3 and T4: affected by changes in thyroxine binding globulin.
- 2- Free t3 and T4: the most accurate.
- 3- Measurement of serum TSH (0.5 micro U/L): differentiates between mild toxicity and anxiety.

B- Radiological:

- 1- U/S of the neck: to know whether cystic or solid nodule,
 - For **cystic nodule** we do aspiration,
 - Criteria of benign cyst by U/S?
 - 1- Smooth wall
 - 2- Clear fluid
 - 3- After aspiration → complete collapse
 - 4- Cytology: no malignant cells.
 - For **solid nodule**
 - FNAB
 - Frozen section
- 2- 2- Thyroid scan: it differentiates between:
 - Hot nodule (excess iodine uptake) → thyrotoxicosis.
 - Warm nodule (normal iodine uptake) → may be functioning adenoma.
 - Cold nodule (low iodine uptake):
 - Malignant (20%), SNG, thyroid cyst, non-functioning adenoma or Hashimoto thyroiditis.

C- Instrumental (biopsy).

II- Pre-operative investigations:

A- Laboratory:

- 1- CBC: RBCs → anemia - WBCs → agranulocytosis.
- 2- FBC: secondary DM may occur.
- 3- KFTs.
- 4- LFTs (hepatotoxicity with antithyroid drugs).

B- Radiological (CXR):

- RSG, calcification, deviation of trachea or metastasis.

C- Instrumental:

- 1- Indirect laryngoscopy: vocal cord mobility, medicolegal purpose.
- 2- Bronchoscopy. 3- Esophagoscopy. 4- ECG.

Thyroglossal cyst

Embryology

- Thyroglossal trunk develops from the foramen caecum of the tongue and descends into the neck → gives thyroid isthmus and medial parts of thyroid lobes.

Etiology

- Unobliterated portion of thyroglossal duct

Site

- At any point of the course of thyroglossal tract, commonest site is subhyoid in the midline.

Symptoms

- Type of patient: child 6-8 years with swelling in the front of the neck
- Painless mass in the midline of the neck (painful if infected).

Signs

- Cystic mass (Paget's test) in midline of the neck moves with deglutition and protrusion of the tongue.

Complications

- Thyroglossal fistula (always acquired & never congenital).
- As any cyst.

Investigations

- U/S → cyst.
- Fistulogram if fistula coexists.

Treatment

- Sistrunk operation (excision of cyst, track and central part of hyoid bone).

Simple Goitre

Definition

- Non-inflammatory, non neoplastic and non toxic enlargement of thyroid gland.

I- Simple Goitre

1. Physiological Goitre

(Venus neck) (diffuse hyperplastic goiter)

- ▶ It may occur in **female at puberty or during pregnancy and lactation**.
- ▶ It is due to ↑ body demand, which will lead to relative iodine deficiency
- ▶ Patient complains of mild enlargement of the gland
- ▶ **Prevention:** use iodized table salt (potassium iodide 1 : 10000)
- ▶ **Treatment:**
 - No treatment (mild cases)
 - Thyroid hormone is given, L- thyroxin 0.2 mg / day for several months to be tapered to 0.1 mg / day for several years.

2. Colloid Goitre

- 1- **Etiology:** is a late stage of diffuse hyperplasia .
- 2- **CIP:** The gland is diffusely enlarged (Soft, Smooth, Symmetrical).
- 3- **Fate:** It may return to normal or cause pressure manifestations.
- 4- **Treatment:** Conservative unless causing pressure manifestations → Subtotal Thyroidectomy.

II- SNG

Incidence

- Female at 30-40 years.

Etiology

- Repeated fluctuations of TSH level → hemorrhage → necrosis → fibrosis → nodules.

Symptoms

- 1- Cosmetic deformity (main complaint)
- 2- Pressure symptoms e.g. :
Pressure on trachea → positional dyspnea and cough especially at night
- 3- Pain: in hemorrhage or malignancy.

Signs

- Inspection
 - Asymmetrical thyroid swelling in lower part of the neck moving up and down with deglutition.
- Palpation:
 - Nodular firm thyroid swelling.
 - Displacement of trachea or tracheomalacia.
 - Shifting of carotid artery pulsation in huge goiter.
- If associated with RSG, there will be:
 - Engorgement of neck veins, lower edge can not be reached, dullness over the manubrium sterni.

Complications

- 1- Pressure on trachea:
 - Unilateral compression → kinking of trachea.
 - Bilateral compression → anteroposterior slit (scabbard trachea).
- 2- Secondary thyrotoxic changes (in 30% of cases)
- 3- Hemorrhage (precipitated by cough or shout):
 - It is an emergency and treated by urgent aspiration or subtotal thyroidectomy.
- 4- Cyst formation: very common
- 5- Infection: very rare.
- 6- Retrosternal extension.
- 7- Calcification.
- 8- Malignant changes → follicular carcinoma (0.5-5%).

Investigations

1- For diagnosis:

- Thyroid function tests: normal T3 and T4 levels.
- U/S of the neck: to detect cystic or solid.
- FNABC: from dominant nodule to exclude malignancy.

2- To exclude complications:

- Thyroid scan: to exclude secondary thyrotoxicosis and retrosternal extension.
- Plain X-ray: to exclude scabbard trachea and calcification.

3- Preoperative investigations: CBC, KFTs, LFTs, FBS and indirect laryngoscopy.

Treatment**1- Treatment of SNG:**

A- Partial thyroidectomy: removal of the nodular part leaving an equivalent of 8 gm of relatively normal thyroid tissue (size of normal lobe) on each side.

- Precautions:

1. We preserve posteromedial part to avoid injury of RLN and parathyroid glands.
2. Postoperative L-thyroxine to avoid recurrence in the left thyroid tissue.

B- Other lines of treatment:

- Total thyroidectomy + replacement therapy to avoid recurrence.
- L-thyroxine: in small-sized swelling in age > 25 years.
- Alcohol injection in the nodules.

2- Treatment of complications:**1. 2ry toxic goitre:**

- If < 45 years → Subtotal thyroidectomy after preparation.
- If > 45 years → Radio-active iodine.

2. Malignant changes (Follicular carcinoma):

- Total or near total thyroidectomy.
- Supplementary L-thyroxine.
- Radioactive iodine for metastasis.

3. Retrosternal extension: surgical excision.**4. Hemorrhage:** urgent aspiration or urgent subtotal thyroidectomy.**Toxic goiter****Definition**

- It is a clinico-pathological condition due to excess thyroxine → ↑ sensitivity of the tissue to circulating adrenaline.

Causes**A- Common types:**

- Primary toxic goiter, secondary toxic goiter, toxic adenoma.

B- Rare causes:

- Neonatal thyrotoxicosis, Hashi toxicosis, De Quervain thyroiditis, thyrotoxicosis factitia, Jod-basedow thyrotoxicosis, functioning secondary carcinoma.

Primary Thyrotoxicosis (Grave's disease)**Definition**

- It is an autoimmune disease that leads to formation of thyroid – stimulating antibodies (IgG)

Pathology

- On stress → exposure of thyroglobulin (sequestered antigen) to immune system → formation of autoantibodies against TSH receptors and have TSH like action → production of thyroxine.

Macroscopic picture

- Diffuse enlargement of the gland

Microscopic picture:

- Proliferation of epithelial lining of the acini.
- The cells are columnar and acini contain less vacuolated colloid

Clinical Picture

- The disease has abrupt onset and shows remissions and exacerbations.

Symptoms

- CNS: irritability, nervousness, insomnia, nightmares and tremors.
- CVS: palpitations, dyspnea (if heart failure)
- Metabolic: ↑ appetite with weight loss and heat intolerance.
- Eye: protrusion of eyeball and diplopia.
- Muscles: easy fatigability.
- GIT: diarrhea or hyperdefecation.
- Urinary: polyuria.
- Sexual:
 - Male: impotence
 - Female: menorrhagia, late amenorrhea.

Signs► **General:**

- Vita signs:
 - a-pulse: Rapid, may be irregular (A.F.), water-hammer pulse.
 - b-blood pressure: high systolic, low diastolic & wide pulse pr.
 - c-temp.: ↑ if thyrotoxic crises.
- Eye: true exophthalmos, eye signs (Dalrymple, Stellwag, Von Graefe's, Mobius & Joffroy's)
- UL: thyroid acropachy, fine tremors, moist warm skin.
- LL: pretibial myxedema, myopathy of proximal muscles.
- Abdomen: H.S.M.

► **Local:**

- Inspection: symmetrical swelling in the front of the neck moves up & down with deglutition.
- Palpation: symmetrical diffuse, firm, non-tender, freely mobile swelling.
- Auscultation: bruit over upper lateral part of the gland.

Investigations**1. For Diagnosis:**

1. **Thyroid function tests:** - Free T3 & T4 → high. - TSH → suppressed.
2. **U/S of the neck:** mild diffuse enlargement.
3. **Thyroid scan:** diffuse ↑ uptake and exclude retrosternal goitre.
4. **Thyroid Antibodies:** TSI.

2. Preoperative Investigations:

1. CBC, LFTs, KFTs, FBS, CXR and ECG.
2. Indirect laryngoscope

Treatment

I- Treatment of Grave's disease

A- Medical treatment (main line of treatment):

- Thiouracil group (inhibit peroxidase and iodine binding to tyrosin):
 - Neomerazole:
 - 10 mg tds → euthyroid → the dose gradually decreased to 5 mg tds for 12-18 months. (S/E: agranulocytosis & hepatotoxicity)
 - Propylthiouracil (used during pregnancy): 100 mg/8 hrs.
- Beta-blockers (propranolol): it protects the heart from arrhythmia.
 - 40mg tds with neomecazone → euthyroid state → stop beta-blocker

B- Surgical treatment: (subtotal thyroidectomy after prepration):

Indicated in: failure of medical treatment, RSG or huge sized goitre.

C- Radioactive iodine (10 millicuri):

Indicated in: failure of medical treatment in patient > 45 years.

II- Treatment of special problems

A- Toxic goiter in pregnancy:

Antithyroid drugs (propylthiouracil) + Beta-blocker + small doses of L-thyroxin in 3rd trim. to suppress maternal TRH to avoid transmitted thiouracil goiter.
(surgical treatment if indicated, better performed in 2nd trimester)

B- Toxic goiter children:

- Antithyroid drugs waiting for spontaneous remission (the ideal)
- If failed → near total thyroidectomy but not preferred

C- True exophthalmos:

1- Treatment of the primary thyrotoxicosis:

Medical:

Antithyroid drugs + small doses of L-thyroxin to suppress TSH and EPF.

Surgical (subtotal thyroidectomy after prep.):

if indicated exophthalmos should be stationary for 6 months.

2- Treatment of exophthalmos:

- Medical:

- a- Head up during sleep, diuretics, prednisone (locally in high doses).
- b- Protection of the eye by day → eye drops and by night → ointment.

- Surgical:

Lateral tarsorrhaphy or orbital deroofing.

	Primary Thyrotoxicosis	Secondary Thyrotoxicosis	Toxic Nodule
Age	Young female (20 – 30)	Middle age & elderly female (30 – 40)	Occur at any age.
On top of	Normal gland	SNG	
Onset	Abrupt	Gradual	Gradual
Course	Exacerbations & remissions.	Slowly progressive.	Slowly progressive
Clinical pic.: Main manifest.	Nervous & metabolic manifestations due to younger age group	CVS manifestation due to older age group	According to age. - Old → CVS - Young → CNS
Pr. Manifest.	Mild or absent	May be present (If large)	Usually absent
Other manifestations	Immunological manifest: - Pretibial myxedema - Clubbing - HSM & Enlarged L.N. (R.E.S. hyperplasia) - <u>True exophthalmos</u>	- NO immunological manifest. - False exophthalmos	False exophthalmos
Thyroid gland	1. Diffusely enlarged & soft with signs of hyper-vascularity 2. Mass occur at the same time of toxicity	1. Nodular & firm 2. Mass occur before toxicity	Single palpable nodule in otherwise normal gland
Treatment			
Medical	<u>Main line of treatment</u> (waiting for spontaneous remission)	Only for preoperative preparation(as the course is progressive)	
Surgical	<u>Indicated in:</u> ▪ Failure of medical treatment (no remission within 1 ½ years) ▪ RSG or huge size goitre	Main treatment	< 45 years old
Radioactive Iodine	> 45 years old with failure of medical treatment	Less effective as fibrosis will prevent penetration so used only in high risk patients (heart failure)	➤ >45 years old ➤ It is very effective but used in old age for fear of malignancy

Medical Treatment		Surgical Treatment		Radioactive Treatment
Carbimazole 10 mg tds	Propranolol 40 mg tds	Preparation		- 10 millicuri of radioactive iodine - 3 months, another dose may be given.
↓		Carbimazole + Propranolol	Rapid Preparation by propranolol for 4days	
		↓		
		Euthyroid state		
		↓		
↓		Carbimazole + Lugol's iodine for 2 weeks		
		↓	↓	
		Subtotal Thyroidectomy (ipsilat. Total lopectomy in toxic nodule)		
		↓	↓	
Carbimazole alone 5 mg tds for 12-18 months		Postoperative propranolol for few days	Postoperative Propranolol for 2 weeks	

Thyroid Gland

Exophthalmos

Definition

- Protrusion of the eye ball due to thyroid disease.

Types

- **False (lid retraction):**
 - Retraction of Muller's muscle due to excess thyroxine.
 - It accompanies both diffuse and nodular goiters.
- **True (actual proptosis)**
 - Actual protrusion of the eye ball.
 - Exophthalmos producing factor (EPF) causes: retrobulbar edema and deposition of fat.
 - It accompanies Grave's disease only.

Complications

- Keratitis, corneal ulceration, panophthalmitis and retinal damage

Examination of exophthalmos

To show true or false: Naffziger's, Frazer's or Ruler's methods.

To determine the degree: exophthamometer or Ruler.

Treatment

⇒ There are 2 parallel lines Should be fulfilled:

1. Treatment of the primary thyrotoxicosis:

Medical: antithyroid drugs + small doses of L-thyroxin to suppress TSH

Surgical: if subtotal thyroidectomy is indicated, exophthalmos should be stationary for 6 months.

2. Treatment of the exophthalmos

Medical

1. Head up during sleep to decrease eye congestion.
2. Protection of the eye by day → eye drops & by night → ointment.
3. Dehydrating measures (diuretics).
4. High doses of prednisone (local administration is risky especially in presence of venous congestion).

Surgical

1. Lateral tarsorrhaphy (it does not prevent the progression).
2. Orbital deroofing (Naffziger operation).

Retrosternal Goitre (RSG)

- More common in males due to strong strap muscles and short neck.
- May be simple, toxic or malignant.

Types

- Plunging: rises with deglutition then descends again.
- Intrathoracic: completely present in chest and separated from the main gland.
- Mediastinal: completely present in the chest but connected with thyroid by band of tissue.

Symptoms

- History of cervical goitre which disappeared with severe pressure symptoms e.g. dyspnea
- Toxic manifestations might be found.

Signs

- **Inspection**: engorgement of neck veins, cyanosis and edema of the face and neck.
- **Palpation**: the lower border is not felt.
- **Percussion**: dullness over the manubrium sterni.

Investigations

- Plain X- ray → soft tissue shadow in superior mediastinum.
- Isotope scan → differentiate between intrathoracic goitre from a mediastinal tumor.

Treatment

- **Subtotal thyroidectomy** from the neck (piece meal) because the vascularity in the neck but if huge or intrathoracic median sternotomy may be needed.
- If **toxic subtotal thyroidectomy after preparation by Inderal** is done but not anti thyroid drugs to avoid the increase of the size of the gland

Autoimmune thyroiditis (Hashimoto's thyroiditis)

Etiology

- Due to auto Ab to thyroglobulin & microsome → Ag-Ab reaction → destruction of thyroid follicles.

Pathology

Macroscopic

Gland is large & nodular due to granulation tissue

Microscopic

Acidophilic **Askanazy cells** + heavy lymphocytic infiltration.

Clinical picture

- Manifestations of thyrotoxicosis in 5% of cases (in early stage)
- Manifestations of myxedema.
- Autoimmune manifestations e.g. erythema nodosum.
- Other autoimmune diseases e.g. vitiligo.
- The gland is asymmetrical large, firm and nodular.

Complications

- Myxedema
- Pressure effects
- Lymphoma

Investigations

- Antimicrosomal antibodies

Treatment

- **Medical** → Cortisone + L- thyroxin.
- **Surgery** → if Large with pressure manifestations Or suspicion of malignancy.

Riedle's thyroiditis

- Extensive fibrosis of the neck, thyroid (**frozen neck**).
- Retro- peritoneal fibrosis, mediastinal fibrosis.
- Treatment → Isthmectomy to relief pressure & to take biopsy.

Subacute Thyroiditis

- Considered to be a viral infection (or as a complication of Mumps).
- Microscopy there is giant cells
- Large tender thyroid gland & febrile reaction
- Hypothyroidism is usually present
- Treatment: → Conservative.

Thyroid neoplasm

- **Benign:** papillary adenoma, follicular adenoma
- **Malignant:**
 - **Primary:** papillary carcinoma, follicular carcinoma, anaplastic carcinoma, Medullary carcinoma, Lymphoma
 - **Secondary:** from direct or blood born spread

	Papillary carcinoma	Follicular carcinoma	Anaplastic carcinoma
Incidence	60%	17%	13%
Age.	Children & young adults	Middle age	Elderly
Sex (F:M)	3.5 : 1	2 : 1	1 : 1.3
Predisposing Factors	1. External irradiation of neck in children which was previously used for TTT of hemangiomas, T.B lymphadenitis. 2. papillary adenoma 3. genetic factors : Godwin \$	1. SNG. 2. Follicular adenoma.	Usually De Novo
Macro	Ill-defined mass infiltrating the surroundings		
	- Grey in color - Multicentric (80%) due to intrathyroid lymphatic spread	- Brown - Unicentric	- Grey - unicentric
Micro	Loss of polarity & signs of mitosis.		
	- Malignant papillae with vascular C.T. core covered by malignant cells - Laminated calcified bodies (psammoma bodies) - orphan annie eyed nuclei	- Thyroid follicles with variable degrees of differentiation - Capsular & vascular invasion	- Clusters of spindle cells (small or large) - Separated by little fibrous tissue
Spread	Mainly lymphatic to deep cervical L.N (Aberrant thyroid)	Mainly blood to <u>skull</u> usually solitary, painful, pulsating, osteolytic (DD. abscess)	Mainly direct & can infiltrate carotid artery which may rupture.
TSH	Dependant	Less dependant	Not dependant

C/P				
A- Typical presentation	Symptoms: - Rapid growing swelling in the front of the neck. - Painless, late painful - Pressure manifestations - Metastatic manifestations			
	Signs: -Thyroid swelling: early mobile, late hard fixed -Neck LNs: may be enlarged and hard -kocher sign: trachea is fixed to gland -berry's sign:absent carotid pulse d.t. infiltration of carotid sheath			
B- Occasional presentation	-Enlarged neck LNs while thyroid carcinoma is not palpable	-Solitary skull metastasis		
Investigations	1-For diagnosis: U/S, FNABC	2-For staging: CT scan, CXR, Bone scan, abdominal U/S	3-Preoperative preparation: CBC, FBS, LFTs, KFTs, ECG	4-For follow-up: *Tumor markers *PET
Prognosis	Good		Bad	
10 years survival	90%		Encapsulated 97% Invasive 70%	
			Very bad	
			Die within 1-2 years	

Treatment

1. **Prophylactic** → avoid predisposing factors as neck irradiation

2. **Treatment of the tumor:**

A- Differentiated carcinoma (Papillary & follicular):

1-Surgical:Total thyroidectomy +block dissection of neck LNs +L-thyroxin replacement (preserve parathyroid).

2-radioactive iodine:to destroy any remnant+ablate any metastasis.

B- Anaplastic carcinoma:

1- The majority of cases are irresectable at time of presentation,so ttt. Is palliative:

- Tracheostomy, isthmus resection or surgical debulking.
- Radiotherapy and chemotherapy.

2-Rare cases are operable → total thyroidectomy followed by radio & chemotherapy.

3. **Treatment of complications:**

- 1) Tracheostomy for tracheal infiltration.
- 2) Gastrostomy for esophageal infiltration.

4. **Post operative follow up:**

- 1) Every 3 month by thyroid scanning, clinical examination and tumor marker.

Prognosis of differentiated thyroid carcinoma

➤ **Certain factors increase the risk:**

- 1- Age. Males above 40 and females above 50 years of age.
- 2- Size of the lesion if more than 5 cm in diameter.
- 3- Presence of capsular or blood vessel invasion

Medullary carcinoma

- **ORIGIN:** From parafollicular **C cells**.
- **Pathology:** It has **amyloid substance** in the stroma
- **E/O:** **sporadic** Or **familial** (MEN II → hyperparathyroidism , pheochromocytoma&medullary C.)
- **C/P:** as be4 +
It is ch.ch. by **Secretion of 5 HT** in the bl. → diarrhea , flushing&bronchospasm(carcinoid \$)
- **Investigations:** as be4+
for screening: in familial type → **Calcitonin** → if high in pt. relative → prophylactic thyroidectomy even if gland is normal (also it is used as a tumor marker).
- **Treatment :**
 - **Thyroid** → Total thyroidectomy & block dissection of LN.
 - **Parathyroid** → if sporadic: preserve all parathyroid glands
If familial: preserve only 1/2 parathyroid gl.

N.B. :

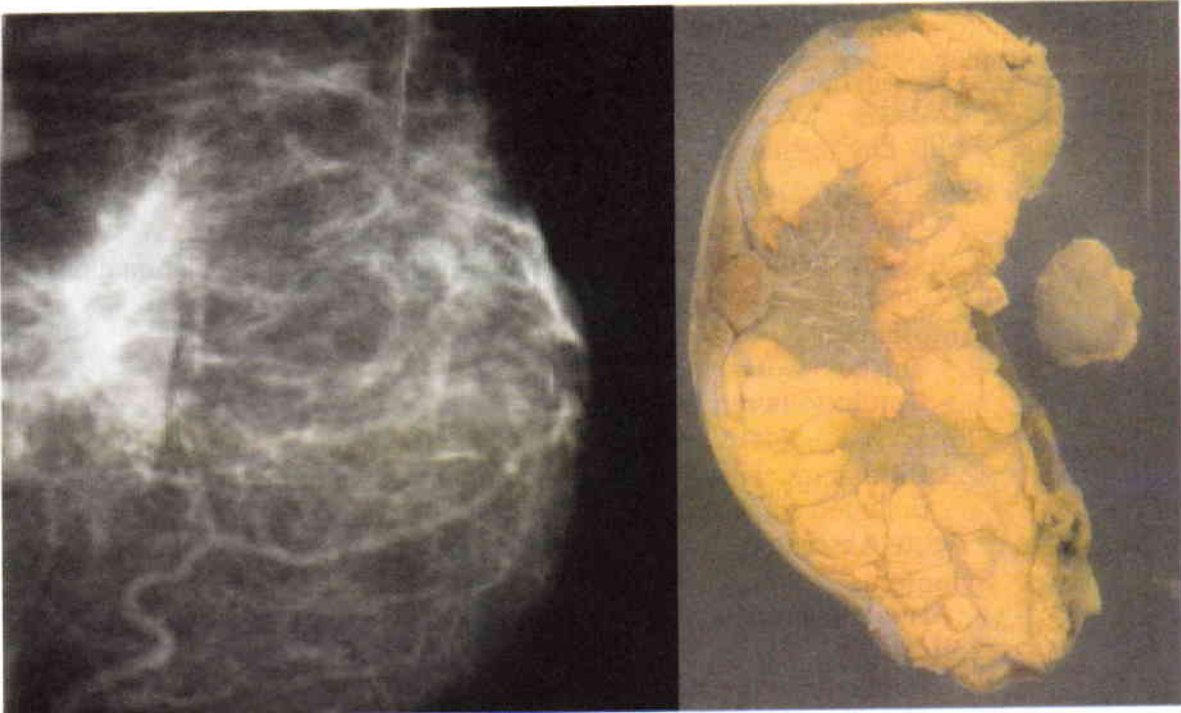
- No rule for radioactive iodine as C cells can't pick up iodine.
- In familial type: treat pheochromocytoma at 1st by combined alfa&beta blockers then adrenalectomy.

Solitary thyroid nodule

➤ See D.D. book.

3

BREAST



Inflammation of the Breast

Acute Lactational Mastitis & Breast Abscess

Definition

- Acute bacterial inflammation of the breast which occurs during lactation.

Etiology

Causative Organism

- **Staph. aureus**

Route of Infection

- **Direct spread (Main route):** from the baby mouth.
- **Blood spread (less common)** from a septic focus.

Predisposing Factors

- **Milk engorgement** (commonest).
- Nipple cracks & fissures caused by suckling
- Retracted nipple.
- Bad hygiene.
- Bad general condition as diabetes, steroid therapy.

Pathology

- Milk engorgement → not treated well → acute mastitis → necrosis by staphylococci → Multilocular Abscess.

Clinical Picture

1. Stage of Milk Engorgement:

⇒ Symptoms:

- Dull aching pain.
- Mild pyrexia.

⇒ Signs: enlargement & induration of the breast (no signs of inflammation).

2. Stage of Acute Mastitis:

⇒ Symptoms:

- The pain worsens
- Continuous high pyrexia

⇒ Signs:

- Diffuse redness, hotness & tenderness of the breast (signs of inflammation)
- Enlarged elastic tender axillary LNs.

3. Stage of Acute Abscess:

⇒ Symptoms:

- Hectic fever, rigors
- Throbbing pain.
- Discharge (pus).

⇒ Signs:

- Hectic fever (i.e. at night it may reach 40° or more due to absorption of toxins due to vasodilatation).
- Edema of the overlying skin + localization of signs of inflam.
- No response to medical treatment. (Persistence of local signs > 5 days or severe systemic upset > 2 days after full antibiotic treatment)
- Fluctuation is a late sign.

(NEVER WAIT FOR FLUCTUATION IN BREAST ABSCESS)

- Enlarged elastic tender axillary LNs.

4. Stage of Chronic Abscess: (See later)

- Attacks of remission & exacerbation
- Tender swelling with yielding center (**Paget's Test**).

Complications

- 1- General: toxemia, septicemia, pyaemia.....
- 2- Local: chronicity.

DD

- 1- **Retromammary abscess**: pain on pushing breast not on bimanual examination.
- 2- **Mastitis carcinomatosis**: low fever with hard fixed LNs.

Investigations TLC, ESR, CRP, C & S.

(It's mainly a clinical diagnosis)

Treatment⇒ **Prophylactic:**a) **During last 2 months of pregnancy:**

- Massage the nipple if not protruding.
- Good hygiene of nipple (Panthinol).

b) **After delivery:**

- Clean nipple after suckling by wet tissue (alcohol free).
- Breast should be completely evacuated with each lactation.

⇒ **Curative:**a) **General Measures:**

- 1- Stop lactation from affected side.
- 2- Lactate with other breast.
- 3- Evacuate the diseased breast manually or by pump.

b) **Medical: (Before abscess development)**

- 1- Antibiotic: Flucloxacillin.
- 2- Analgesic & antipyretic.
- 3- Hot fomentation.

c) **Surgical (incision and drainage):** do not wait for fluctuation.

Through radial incision not reaching areola or circumareolar incision (for better cosmesis) under general anesthesia and destroy all loculi by finger to form single cavity then drainage from most dependent area.

Chronic Breast Abscess

Causes

- Inadequate treatment of acute abscess.
- Persistence of predisposing factors (i.e. Lactation).
- Bad General condition of the patient.

Pathology

- Excess fibrosis with sterile pus.

Clinical Picture

- ⇒ **Type of patient:** history of maltreated abscess.
- ⇒ **Symptoms:**
 - Attacks of remission & exacerbation.
 - Breast mass: may be associated with nipple retraction or skin puckering
 - Pus discharge per nipple.
- ⇒ **Signs:**
 - Slightly tender breast swelling with yielding center (Paget test).
 - Axillary LNs. are enlarged elastic, tender & mobile.

DD

- 1) Fibro-adenoma.
- 2) Breast carcinoma.

Investigations

- U/S (cyst) & needle aspiration (reveals pus and to exclude carcinoma).

Treatment

- ⇒ **Prophylactic Treatment:** adequate drainage of acute abscess.
- ⇒ **Curative Treatment:** excision.

Mammary Duct Ectasia (Plasma Cell Mastitis)

Definition

- It is chronic non-specific inflammation of major milk ducts.

Etiology

- Unknown.

Pathology

- Dilated ducts, fibrosis and greenish discharge.
- The main cell is plasma cell in inflammatory reaction

Clinical Picture

Type of patient

- Middle-aged female
- More common in **smokers**.

Symptoms

- May be **asymptomatic** or presenting by one of the following:
 1. **Nipple discharge:** white curdy
(Duct ectasia is the most common cause of nipple discharge)
 2. **Subareolar painless** or painful **swelling** if an abscess develops

Signs

- The affected area may be hard with skin dimpling & retraction of nipple (Fibrosis)

DD

- Breast carcinoma.

Investigations

- Subareolar mass: triple assessment.
- Discharge: Benzedine test and cytological examination.

Treatment

- 1) Excision of the major duct through a circum-areolar incision (**Hadfield's operation**).
- 2) Correction of nipple inversion.
- 3) Antibiotics and cessation of smoking may be tried.

Fibroadenosis (ANDI)**Definition**

- Aberration of normal changes occurring during evolution and involution of a healthy breast.

Incidence

- The most frequent breast disorder (it occurs after puberty and before menopause).

Cause (*Unknown*)**Pathology**

- ⇒ The upper outer quadrant of the breast is the commonest site of affection.
- ⇒ The condition is not pre-cancerous.
- ⇒ **It is characterized by:**
 - 1- Adenosis
 - 2- Fibrosis
 - 3- Epitheliosis.
 - 4- Papillomatosis
 - 5- Cyst formation
 - The cyst might be:
 - Microcyst.
 - Large macrocyst. (**blue domed Cyst of Bloodgood**): A large cyst contains altered blood.

Clinical Picture⇒ **Type of patient:**

- It occurs after puberty or before menopause.

⇒ **Symptoms:**

- It may be completely **asymptomatic**.
 - These changes are usually **CYCLIC**.
- 1- **Breast pain (Mastalgia, Mastodynia):**
 - Exaggerated pre-menstrual tension
 - Dull aching or stitching pain
 - 2- **Breast lump (Cysts or sclerosing adenosis):** the most frequent complaint.
 - 3- **Breast discharge:** clear, greenish or brownish.

⇒ **Local Examination:****1- Breast Lump:**

- Commonly bilateral small painful lumps that ↑ at time of menses.
- **Felt by tip of fingers not by flat of the hand.**

2- Discharge.**3- Axillary LNs:**

- May be elastic, enlarged, tender and mobile.

Complications

1. If marked epitheliosis and papillomatosis increased risk of malignancy (1- 1.5 times and atypical hyperplasia 5 times)
2. Hemorrhage and infection in cyst of Bloodgood.
3. Anxiety (if severe pain).

DD

- *Other causes of breast pain and breast lump and discharge.*

Investigations

- **For lump: triple assessment (to rule out cancer):** U/S, mammography and FNABC
- **For discharge:** Benzedine test and cytological examination.

Treatment*Reassurance from cancer phobia***A. Change life-style:**

1. Firm bra.
2. Avoid coffee, tea and chocolate.

B. Medical treatment:

1. Analgesic.
2. Prim-rose oil single evening dose.
3. Parlodel 2.5 mg tab/twice /day (anti prolactin).
4. Danazol tab. (last line of TTT as it causes acne & hirtutism)
5. Psychotherapy.

C. Indication of the surgery:

1. **Biopsy** → if doubtful diagnosis.
2. **Excision of the cyst** → large cyst (cyst of Bloodgood).
3. Cysts are treated by aspiration; recurring cysts are excised for biopsy.

Prognosis

It is not precancerous except if there is marked papillomatosis and epitheliosis.

Breast Neoplasm

I- Benign Tumors of the Breast

Duct Papilloma

Incidence

- The most common cause of bleeding per nipple.

Pathology

- **Site:** usually situated in one of the main ducts near the nipple.
- **Macroscopic:** pedunculated mass bleeds easily.
- **Microscopic:** vascular CT core covered by epithelium.

Clinical Picture

Type of Patient

- 30-40 years female with **bleeding per nipple**.

Symptoms

1. Blood stained nipple discharge. (Commonest symptom).
2. Painless swelling → retention cyst.

Signs

1. **Bleeding per nipple:**
 - By pressure on the swelling.
 - If there is no palpable swelling, **zonal pressure** will reveal the discharge.
2. **Swelling:** may be present.
3. **Axillary LNs:** are **not** enlarged.

DD

- Of other causes of bleeding per nipple.

Investigations

- 1) **Benzidine test** → to make sure is it blood or not.
- 2) **Galactography** the papilloma appears as a regular filling defect.
- 3) **Mammography** → to screen the rest of the breast and the other breast.

Treatment

- It's a **pre-cancerous**, so the treatment is:
 1. **Micro-dochectomy** with 2.5 cm safety margin.
 2. Histopathology.

Fibroadenoma

Incidence

Fibroadenoma is the commonest cause of breast mass in young females
(The usual age is 15-30 years)

Pathology

- Fibroadenoma is a **benign** neoplasm of the breast that affects both fibrous & glandular tissues but **fibrous element predominates**.

Types of Fibroadenoma

Peri-canalicular (Hard)	Intra-canalicular (Soft)
Macroscopic picture: Size: small Surface: smooth. Color: whitish Consistency: firm or hard. Cut section: whorly appearance. Capsule: 2 capsules true and false capsule of compressed surrounding T.	Macroscopic picture: Size: large Surface: lobulated. Color: whitish Consistency: soft Cut section: might show central necrosis Capsule: incomplete capsule.
Microscopic picture: - Formed mainly of fibrous tissue - Fibrous tissue proliferation occurs around the acini & ducts.	Microscopic picture: - Contains more glands - Fibrous tissue proliferation invaginates the ducts.
Complication: Never turn malignant.	Complication: Liable to turn to sarcoma.

Clinical Picture

Type of Patient

- Hard fibroadeonoma 20 – 30 years female.
- Soft fibroadenoma 30-50 years female.

Symptoms

- Painless lump that is discovered accidentally.

Signs

- Breast swelling:**
 - Peri-canalicular:**
 - Usually small, non tender, firm, well-circumscribed with smooth surface & with **high mobility** in breast tissue (**breast mouse**).
 - Intra-canalicular:** may reach huge size, non tender, soft, lobulated with restricted mobility in the breast.
- Axillary LNs:** not enlarged.

DD

(Precancerous lesions)

- Breast carcinoma.
- Duct ectasia.
- Localized fibroadenosis.

Investigations

Clinical picture is usually enough for diagnosis

- Mammography → reveals well-circumscribed lesion.
- U/S → may be needed.

Treatment

- **For peri-canalicular:** enucleation through circum-areolar incision
- **For intra-canalicular:**
 - If Small → Excision with safety margin.
 - If large (Cystosarcoma phylloides) → wide local excision (to prevent recurrence) or if the tumor is occupying the whole breast → simple mastectomy.

II- Malignant Tumors of the Breast

Breast Cancer

Age

- Mean age of affection is 60 years.

Sex

- 99.5% in female. - 0.5% in male.

Predisposing Factors

1. **Genetic Factors:** BRCA-I and II, P53
2. **Endocrinal factors:** early menarche & Late menopause
3. **Precancerous lesions** duct papilloma.
4. **Obesity & ↑ fat intake**
5. **Race:** more in white women than Asian or Africans
6. **Radiation therapy to the chest**

Classification

A- Paget disease of the nipple.

B- Duct carcinoma:

- 1- Non-infiltrating: comedo type, solid and papillary.
- 2- Infiltrating: scirrhous, medullary carcinoma, mastitis carcinomatosis and mucinous (colloid).

C- Lobular carcinoma: infiltrating and non-infiltrating.

Spread of Cancer Breast

A. Direct Spread:

1. Breast tissue.
2. Skin.
3. Pectoral fascia.
4. Pectoralis major.
5. Serratus anterior.
6. Chest wall.

B. Lymphatic Spread:

(By both embolization & permeation)

1. Axillary LNs (common)
2. Internal mammary LNs (next common)
3. Supraclavicular LNs (in advanced disease)

C. Blood Stream Spread:**D. Transcelomic Spread:***(After liver affection)*

1. Ovaries → **Krukenburg's tumor** (by retrograde lymphatic spread)
2. Douglas pouch
3. Peritoneum → **malignant ascites**.

Staging of Cancer Breast

Manchester Staging (Better in Clinical)

Stage I

- Mobile mass in the breast.
- Not attached to pectoral muscles or chest wall.
- No palpable axillary LNs.

Stage II

- Mobile mass in the breast
- With or without skin teathering (in area **less** than tumor periphery).
- **Not attached** to pectoral muscles or chest wall.
- **Palpable mobile ipsilateral axillary** LNs.

Stage III (stage of wide local spread)*Any of the following:*

- Skin affection.
- **Fixed to pectoral muscles**.
- **Fixed Ipsilateral axillary** LNs.
- **Ipsilateral supra clavicular** LNs affection.
- **Ipsilat. Edema of the arm**.

Stage IV

- Skin affection **wide** of the breast (**Cancer en cuirasse**).
- **Fixed to chest wall**.
- Involvement of **opposite breast or axilla**.
- **Distant metastasis**.

Staging of the UICC (Union Internationally Contre Cancer)

➤ It is based on the TNM staging:

Stage UICC	Description	category	5 year survival (%)
I	T1,N0,M0	Early breast cancer	93
II	IIA T2,N1,M0	Early breast cancer	72
	IIB T3,N0,M0		
III	IIIA T1-3,N0-2,M0	Locally advanced breast cancer	41
	IIIB T4,anyN,M0		
IV	Any T, any N ,M1	Metastatic	18

Management of early breast cancer

Definition

- Early breast cancer is up to stage I, II in Manchester staging.

Clinical picture

- **Symptoms:**

- Painless swelling.
- Negative occult presentations

- **General signs:** negative

- **Local signs:**

- Inspection:**

- 1- Breast enlargement and asymmetry.
 - 2- Skin lesions:
 - a- Nipple retraction and skin dimpling.
 - b- Peau de'range

- Palpation**

- 1- Firm to hard mass freely mobile.
 - 2- Axillary LNs: negative or may be enlarged (hard and mobile)

DD

- Other causes of breast mass.

Investigations

- For diagnosis: Triple assessment.
 - 1- Clinical 2- Mammography. 3- Histopathology and cytology.
- For staging: CXR, bone scan, LFTs
- Preoperative organ profile.

Treatment

- **Prevention:** early detection through.

- 1- Self assessment.
 - 2- Physical examination.
 - 3- Mammography.

- **Definitive treatment:**

- Our aim is cure

- 1- Local:**

- a- Surgery:**

- i- Radical: modified radical mastectomy with reconstructive surgery by myocutaneous flap or silicon implants either immediate or delayed..
 - ii- Conservative surgery: mostly done:
 - Lumpectomy with axillary surgery or radical dose of radiotherapy.
 - Advantages: less psychological morbidity.

- b- Radiotherapy:**

- Postoperative after conservative surgery by radical dose (5000 RAD) or after radical surgery by adjuvant dose (1500 RAD).

2- Systemic:

- Prolong survival, ↓ recurrence and treat micrometastasis.
 - a- Hormonal therapy: if hormone receptor +ve we use selective estrogen receptor modulators as Tamoxifen
 - b- Chemotherapy: by CMF regimen, in all patients with LNs +ve or -ve but with poor prognostic criteria.
- ☒ **Paget's Disease**
 - Treated by modified radical mastectomy.
- ☒ **Carcinoma in situ**
 - If lobular → no treatment
 - Ductal → local excision with follow-up
- ☒ **Cancer breast with pregnancy**
 - Modified radical mastectomy, no radiotherapy, chemotherapy after second trimester.

■ **Treatment of complications****Intermediate (Locally advanced) breast cancer**▶ **This category of patients have:**

- ☒ This category of patient have cancer above 5 cm diameter.
- ☒ Fixed axillary L.N.s or internal mammary L.N.s.

▶ **For this:**

- ☒ Distant metastasis should be excluded by C.T. scan & PET scan.

▶ **Treatment:**

- Neoadjuvant chemotherapy → down staging:
 - If good response → breast conservative therapy.
 - If poor response → modified radical mastectomy.

Management of Advanced Breast Cancer**Definition**

- This category includes stages iii and vi uicc, t4 patients, fungation, ulceration, inflammatory carcinoma or recurrent cases as well as distant metastases.

Clinical picture▶ **Symptoms**

- Painful swelling
- Positive occult presentations

▶ **General signs**

- May be positive for evidence of metastasis

▶ **Local signs****Inspection**

1. Breast enlargement and asymmetry.
2. Skin lesions:
 - a) Skin nodules.
 - b) Sister joesph nodules.
 - c) Cancer en cuirasse
 - d) Skin ulceration.
 - e) Brawny edema

Palpation

- 1- Hard fixed mass.
- 2- Axillary LNs: hard and fixed.

DD

- Other causes of breast mass.

Investigations

- For diagnosis: Triple assessment.
1- Clinical 2- Mammography. 3- Histopathology and cytology.
- For staging: CXR, bone scan, LFTs
- Preoperative organ profile.

Treatment**A. Primary treatment:****I- Hormonal therapy:**

- Only given to hormone receptor +ve patients.
- More effective in → post-menopausal women.
- Not effective in → hormone -ve patients, visceral metastasis & young patients below 35 yrs.
- Tamoxifen is given → if no response → use aromatase inhibitors.

II- Chemotherapy:

- It is the basic treatment of metastatic diseases.
- **Indications:**
 1. Rapidly progressive disease.
 2. Pre-menopausal women.
 3. Visceral metastasis.
 4. Hormone receptor -ve cases

- **Combination:** CMF

B. Adjuvant treatment:**1- Radiotherapy:**

- Palliative in:
 - a. Pain (bone metastasis).
 - b. SVC obstruction.
 - c. Used to control tumor fungation.

2- Surgical treatment:

- a. Modified radical mastectomy in stage III.
- b. Simple mastectomy in stage IV.

C. Treatment of complications

- e.g. external irradiation + internal fixation of pathological fractures.

Follow-up of Patients with Breast Cancer

⇒ After treatment, every 3 months for first 2 yrs, then every 4 months next 2 yrs, then yearly for life. For:

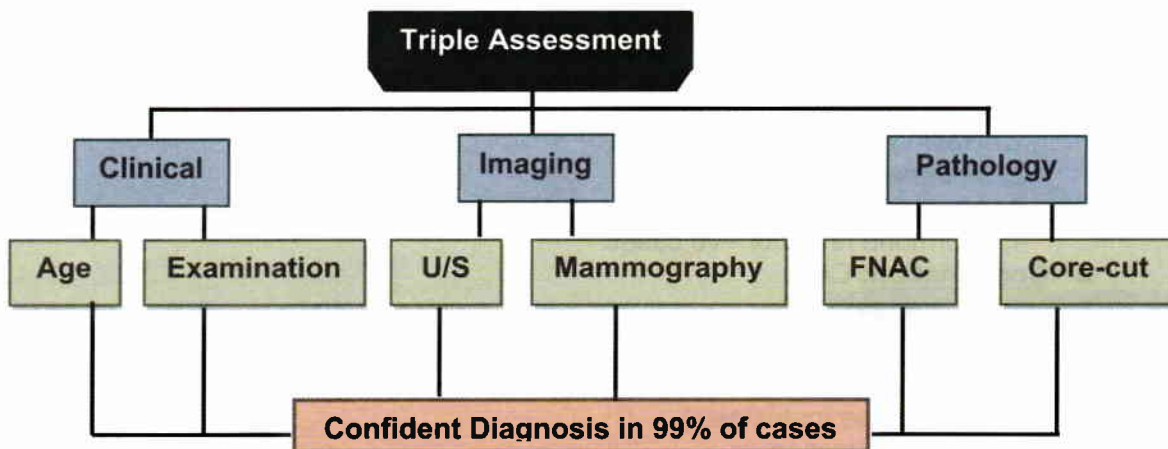
1. Detect and treat complications of mastectomy as:

- Psychiatric morbidity caused by the loss of the breast
- Arm edema results from excision of lymphatics or their obstruction by radiotherapy.

2. Detect local recurrence or distant disease.

Prognostic Factors of Breast Cancer

1. **Type of the tumour:** best with in situ carcinoma, and paget's disease, worst in the inflammatory carcinoma
2. **The T stage** of the primary tumour. The higher the T stage, the worse is the prognosis.
3. Size, mobility, number, and location of the involved lymph nodes.
 - Large fixed nodes are of bad prognosis
 - The number of involved nodes largely affects the prognosis
 - Negative axillary nodes → 10 years survival rate 65%
 - 1-3 positive axillary nodes → 10 years survival rate of 38%
 - More than 4 +ve axillary nodes → 10 years survival rate of 13%
 - The prognosis worsens the higher the affected nodes in the axilla.
4. Presence of distant metastasis markedly worsens the prognosis
5. Hormone receptor status: +ve receptors tumour has better prognosis.
6. The site of the tumour. Medial half tumour have a worse prognosis than those of lateral half due to early involvement of the internal mammary lymph nodes



- If the 3 parameters are concordant, the surgeon can rely on the diagnosis
- If the 3 parameters are not concordant, a further investigations eg. excision biopsy is needed

Diseases of Male Breast

Cancer Breast in Male

- It's a rare disease.
- Painless lump beneath the Areola at 50 years.
- Bad prognosis.
- More with BRCA II gene

Gynecomastia

- Painless enlargement of male breast due to ↑ glandular elements ≠ Lipomastia.

Causes

- Idiopathic.
- Klinefelter's Syndrome.
- Testicular atrophy.
- Digitalis, estrogen, tagamet, aldactone.
- Late seminoma & teratoma.
- Liver cirrhosis
- Sertoli cell tumor.

Treatment

- 2ry → cause
- 1ry → subcutaneous mastectomy

Management of Breast Lump

► See D.D. book.

Management of Breast Pain

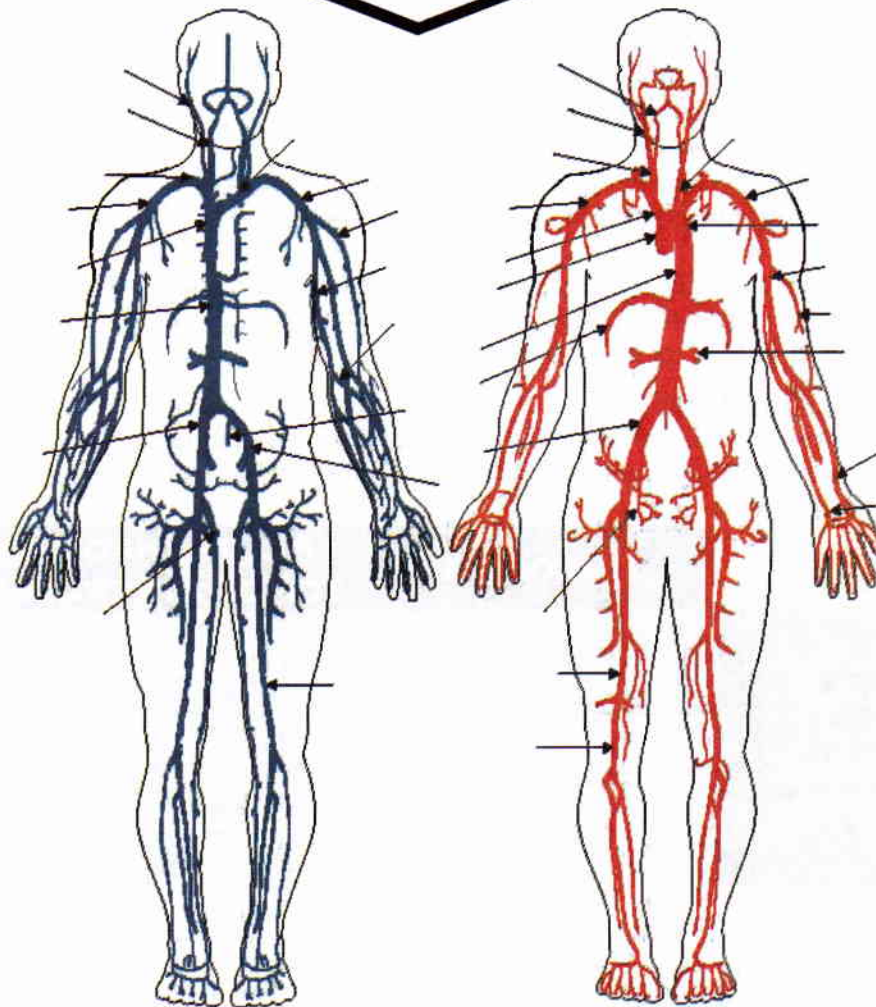
► See D.D. book.

Management of Nipple Discharge

► See D.D. book.

4

VASCULAR SURGERY



ARTERIAL SYSTEM

General Scheme of Acute Ischemia

Definition

- Lack of blood flow due to sudden occlusion of a previously patent artery with **no** time for collaterals to open.

Causes

- 1) Embolism.
- 2) Thrombosis.
- 3) Trauma e.g. catheter, arteriography. . Intra-arterial drug injection
- 4) Others (Pressure from outside e.g. tournique, plaster cast, aortic dissection, phlegmasia Alba dolens).

Clinical Picture

⇒ C/P of acute ischemia:

- Onset:
 - Sudden in embolism.
 - Less dramatic in thrombus.
 - Following trauma in arterial injury.
- 6Ps:
 1. Pain: sudden severe.
 2. Pallor: replaced later on by mottled cyanosis.
 3. Paralysis.
 4. Parasthesia (Bad sign): usually progress to frank anaesthesia.
 5. Pulselessness : distal to site of obstruction.
 6. Progressive coldness of the limb.

⇒ C/P of the cause:

- Embolism: A.F, other system affection (painless hematuria, hemiplegia, or MVO).
- Thrombosis (intermittent claudication, other system affection e.g.: IHDS).
- History of trauma.

	EMBOLISM	ACUTE THROMBOSIS ON TOP OF ATHEROSCLEROSIS
History	Arrhythmia or recent M.I	claudication
Source of emboli	Usually present	absent
Radial pulse	Usually irregular (AF)	Usually regular
Skin colour	white	dusky
Limb nutrition	normal	Picture of chronic ischemia
Angiography	Sharp cut-off Minimal collaterals	Tapering stenosis Diffuse atherosclerosis Extensive collaterals

Classification

- 1- Viable
- 2- Threatened
- 3- Irreversible
 - Fixed color changes (blue staining).
 - Signs caused by muscle necrosis (tense calf, fixed planter flexion, bulging anterior leg compartment).
 - Acute paraplegia may occur in saddle aortic aneurysm.

Pathological consequences

- 1- Secondary arterial thrombosis.
- 2- Compartmental syndrome. Early fasciotomy of the muscles compartments can avoid this problem.
- 3- As a result of stasis of blood in the arterial tree, blood stagnates in the veins draining the limb and DVT may develop.

Complications

- Wet gangrene.
- DVT.
- Nerve ischemia → impairment of conduction.
- Chronic ischemia after complete recovery.

DD

From other causes of acute limb pain (see the textbook)

Investigations

► Investigations should be urgent.

A. Imaging studies:

- 1- Duplex scan: localizes and identifies the presence of embolism and thrombus.
- 2- Arteriography: may cause a delay of 2-3 hours. It is, therefore, not done in a threatened limb. Its value is in cases of diagnosed acute thrombosis because it provides information that is essential before doing an arterial reconstruction. This information includes:
 - a- Site of occlusion.
 - b- Proximal inflow, i.e., if there is another proximal arterial narrowing.
 - c- Distal run-off, i.e., flow distal to the obstruction.
- 3- Echocardiography detects cardiac sources of emboli.

B. Laboratory studies:

- 1- ↑ hemoglobin, BUN and creatinine indicate intravascular hypovolemia due to fluid sequestration in the limb.
- 2- Acidosis and r↑CPK and WBCs indicate extensive muscle necrosis.

Treatment

■ Preoperative preparations:

- **Morphine:** for pain.
- Prophylactic **antibiotics**.
- **Heparin:**
 - Deal with cardiac problems.
 - Decrease propagation of distal thrombosis.
- Care of the **cardiac** condition. (O₂, Digitalis).

■ Operation:

• Embolism:

- Urgent embolectomy (within 6 hours)
by Fogarty catheter+ Fasciotomy. "*Done in late cases as a prophylaxis to prevent compartmental syndrome*".
- Followed by anticoagulant .

• Acute Thrombosis on top of chronic:

- If viable→ Thrombolytics followed by revascularization surgery.
- If threatened→urgent arteriography followed by urgent revascularization surgery.
- If irreversible→amputation.

• Arterial injury:

☞ First aid:

- ▶ Control external bleeding(if present).
- ▶ Anti shock measures.

☞ Definitive treatment (within 6 hours):

- ▶ if open→immediate exploration &repair.
- ▶ if closed→Reduction of fracture if present:
 - If pulse returns → follow up.
 - If no pulse (within 20 min) → explore and repair according to type of injury:
 - Spasm → paint the artery with papaverine.
→if failed: dilate using fogerty catheter.
→if failed:Excision & graft .
 - contusion→excision of contused segment&vein graft.
 - Partial tear:
 - if<1/2 circumference:direct suture or venous patch.
 - if 1/2 circumference:turn it complete&deal with it as complete tear.
 - Complete tear:
 - if no gap:direct suture in oblique line to avoid annular stricture.
 - if there is gap:•mobilize artery by dissection.
 - If failed→cut branches that prevent mobility.
 - If failed→saphenous graft.

■ Post operative:

• Treatment of the cause:

- Embolism →Antiarrhythmic drugs if AF, exclusion graft if aneurysm.
- Thrombosis →conservative treatment of chronic ischemia.

• Treatment of complications:

- Gangrene →amputation.
- Volkmann's ischemic contracture →Muscle or tendon transfer .
- Crush syndrome →I.V fluids + alkalinization of urine .

Arterial Embolism

Definition

Sudden impaction of an embolus (foreign body to the blood stream) in a blood vessel causing obstruction.

Etiology (sources of emboli)

1. **From the heart (most common):**
 - Lt. atrium: Mitral stenosis with AF{the commonest} (77%) <. atrial myxoma
 - Lt. ventricle: mural thrombus.
 - Valves: Sub acute bacterial endocarditis.
2. **From large arteries** (i.e. arterioarterial immobilization).
 - Atherosclerosis.
 - Aneurysms.
3. **From veins:**
 - Venous emboli through patent VSD or ASD + Eisenmenger → Paradoxical emboli.
4. **Other types:** air embolism, fat embolism, therapeutic embolization (in TTT of tumors & bleeding) & amniotic fluid embolism.
5. **5% unknown cause.**

Pathology

1. Impaction occurs at site of branching (as arteries gets narrower).
2. **Immediate cut off of the blood supply** → severe ischemia + VC of collaterals (reflex due to severe pain).
3. **Muscle necrosis** within 4 -6 hours.
4. **2ry thrombosis** distal to the impacted embolus and proximal till the 1st proximal branch due to stasis of the blood in this region , but from the heart to the last branch proximal to embolus circulation is good .
5. **DVT.**
6. Compartmental syndrome (if prolonged ischemia).

Site

- According to incidence (from most to least common):
 - Femoral. - Aorta. - Popliteal.
 - Brachial. - Common carotid.

Clinical Picture

- See the general scheme of acute ischemia.

Complications

- See the general scheme of acute ischemia.

DD

1. **Causes of acute limb pain:** CB4.
2. **Of the cause** Embolus x Thrombus جدول.

Investigations

A. Site of impaction:

- **Doppler U/S, Duplex:** absent blood, flow distal to site of occlusion.
- **Angiography:**
 - Block of the main arterial tree .
 - With **no or minimal collaterals**.

B. Detection of the cause: ECG, Echo.

C. detection of complications: muscle necrosis → ↑TLC, ↑CPK

Treatment

- See the general scheme of acute ischemia.

Acute Arterial Thrombosis

Definition

- **Thrombosis** is the formation of a clot or thrombus inside a blood vessel, obstructing the flow of blood.

Etiology

1. **Age** → old.
2. **Predisposing factors** → **diseased wall:**
e.g. Atherosclerosis (commonest cause)..aortic aneurysm or traumatic contusion.
3. **Precipitating factors** → **stasis & hyperviscosity:**
 - Dehydration (diarrhea).
 - Blood disease (polycythemia).

Pathology

- It is acute on top of chronic ischemia due to atherosclerosis (fibrofatty streaks, elevated plaques).
- Collaterals are opened → ischemia is less severe.

Clinical Picture

- See the general scheme of acute ischemia.

Complications

- See the general scheme of acute ischemia.

DD

- Embolus x Thrombus جدول

Investigations

A. Site of impaction → CB4 +angio: block of main arterial tree with appearance of collaterals.

B. Detection of the cause → CBC, lipid profile in blood, blood .sugar, ECG.

C. Detection of complications: see be4.

Treatment

- See the general scheme of acute ischemia.

Arterial Injury

Etiology

- **Penetrating injury** (high velocity ,or low velocity).
- **Blunt injuries** .e.g car accidents,bone fracture.
- **Iatrogenic** e.g.
 - o Intra-arterial drug injection.
 - o Catheterization for the heart (most common femoral).
 - o Angiography.

Pathology (types)

- **With tear**
 - It may lead to:A-V fistula , false aneurysm .
 - Tear may be:
 - a) Complete → retraction of ends →less bleeding .
 - b) Incomplete →more bleeding due to retraction of ends leads to widening of the gap.
- **Without tear:**(present with ischemia)
 - Spasm.
 - Contusion.

Clinical Picture

- **History of the cause:** trauma, bleeding (if external).
- **General examination** →shock , associated injuries .
- **Local examinations** →
 - **Hard signs:** these are sure signs of arterial injury.
 1. External bleeding (pulsatile hemorrhage).
 2. Loss of distal pulsations.
 3. 6Ps.
 4. Pulsating or expanding hematoma.
 5. Palpable thrill or audible bruit heard at or distal to area of injury.
 - **Soft signs:** these are less specific signs
 1. Small or moderate sized. hematoma that is not pulsating and not expanding.
 2. Proximity of penetrating wound to a major vascular structure.
 3. Adjacent nerve injury producing neurological deficit.
 4. History of pre hospital hemorrhage that has stopped or presentation with shock that cannot be explained by other injuries.
 5. Delayed capillary refilling time.

Closed injury is more dangerous due to missed diagnosis.

Complications

1. **Acute ischemia.**
2. **Haematoma .**
 - Injury to artery → pulsating hematoma → false traumatic aneurysm (the wall is fibrous tissue & not normal vessel wall).
 - Injury to artery & vein → A-V fistula → varicose veins & ischemia).
3. **Muscle affection .**
 - Wet gangrene.
 - Volkmann's ischemic contracture

Investigations

- **If Diagnosis established** no need for investigations. Proceed to urgent exploration.
- **If Doubtful diagnosis:** urgent investigations
 - Doppler.
 - Angiography (most accurate) → A traumatic A-V fistula shows early venous filling
 - X-ray for associated fractures .

Treatment

- See the general scheme of acute ischemia

Intra-arterial drug injection

- ▶ The **most** commonly punctured arteries are the brachial and radial arteries.

Clinical features

- 1- Burning discomfort extending from the point of injection to the tips of fingers immediately after injection. This followed by severe pain and blanching.
- 2- Edema develops rapidly and may be severe.
- 3- Coldness and cyanosis.
- 4- The distal pulses are often normal.
- 5- Digital gangrene and less commonly total extremity gangrene.
- 6- Arteriography should not be done.

Treatment

- ▶ **Treatment should start immediately.**
 - 1- Heparin 5000 IU I.V followed by constant infusion.
 - 2- Dexametgazone 4mg I.V every 6 hours.
 - 3- Low molecular weight dextran minimizes platelet aggregation.
 - 4- Strong analgesics.
 - 5- Limb elevation to decrease edema.
 - 6- Fasciotomy is frequently needed due to chemical myopathy.

Chronic Ischemia

Definition

- Gradual cessation of blood supply due to partial or incomplete arterial occlusion with collaterals.

Etiology

- Patient >45 years → Atherosclerosis The most common cause
- Patient <45 years
 - If diabetic → presenile atherosclerosis .
 - If non diabetic:
 - Male: Burger's , Arteritis .
 - Female : Raynaud's , Arteritis .

Clinical Picture

I. C/P of chronic ischemia (may be asymptomatic)

⇒ Symptoms

I. Pain:

Claudication pain

- cause → ms. Ischemia.
- Character → Cramp like
- Site

Aortic	→ from buttocks	downwards.
Iliac	→ from thigh	downwards.
Femoral	→ from calf	downwards.
- Increased by → exercise.
- Relieved by → rest.
- Claudication distance → gradually shorten.
- Period of rest → gradually ↑

Rest pain

- Cause → Ischemic neuritis
- Time → more at night
- Character → severe pain in the dorsum of foot.

Rest pain + color changes = pregangrene.

II. (Associated other systems)

CVS → H.F. & angina pectoris.

CNS → Loss of memory, fainting, blindness or hemiparesis.

Kidney (atherosclerotic kidney) → loin Pain & haematuria.

Genital → Impotence in males .

(Leriche syndrome = aortobi-iliac obstruction).

GIT → Cramping pain in the abdomen in relation to meal.
(Post-cibal abdominal angina).

- Pulse and state of wall.
- Aneurysm (due to atherosclerosis).
- Anemia, chest infection.
- A-V fistula.

1. Skin

- Cold sensation.
- Trophic changes:
 - Loss of hair (1st Trophic change).
 - Thin, atrophic, stretched, dry skin.
 - Ulcer resistant for healing(bet. Toes,on maleoli or dorsum of foot) .
- Color changes: Pale →bluish →blackish.

3. Subcutaneous tissues ↓ limb circumference Thin tapering toes.

Gangrene. (usually **dry** unless with H.F. or infection).

- **Sensory** → Paraesthesia, Hypo or hyperaesthesia
(Proprioception is the first to be lost).

- **Motor** → weakness.
- **Reflexes** → weak ankle ($S_{1,2}$) & knee reflexes ($L_{3,4}$).

- DVT (stasis).
- Migrating superficial thrombophlebitis → in Burger's only.

- a - Weak or absent peripheral pulsations according to level.
- b - Auscultation:-
 - Aneurysm → Systolic bruit.
 - Stenosed artery → Systolic bruit.
 - A-V fistula → Continuous machinery bruit.
 - c-rigid arterial wall(in atherosclerosis).

2- Clinical Picture according to the cause:

	Atherosclerosis	Burger's
Age	> 50 years	20 - 40 years
Sex	Male	
PF	- DM - Obesity -HTN	Smoking
Pathology	Degenerative diseases of arterial wall	Inflammatory disease of neuro-vascular bundle
C / P	1. Progressive 2. Affecting proximal vessels 3. Neuritis is late	1. Fluctuating in course 2. Affecting distal vessels 3. Neuritis is early 4. Thrombophlebitis
Treatment	Direct arterial surgery	Sympathectomy (no run off)

N.B: critical limb ischemia is diagnosed by Persistent rest pain, persistent colour changes, presence of ischemic ulcers or small gangrenous patches.

NB: clinical staging of chronic limb ischemia:

- Stage I: asymptomatic.
- Stage II: intermittent claudication.
- Stage III: rest pain.
- Stage IV: ulceration or gangrene.

Investigation of Chronic Ischemia

⇒ For diagnosis:

1- **Doppler, Duplex: (Non-invasive but less accurate):**

▪ It detects

- Main stem flow, collaterals,
- ABI (ankle brachial index)
 - Normally A/B index is 1-1.2
 - Less than 0.9 denotes ischemia
 - Less than 0.7 indicates severe ischemia
 - Less than 0.3 indicates impending gangrene

2- **Arterial angiography (invasive but accurate):by seldinger needle**

- It is done preoperative to detect site of the vessel wall, the collaterals and run in and run off.

3- **MRA (non-invasive and accurate but expensive and only diagnostic)**

⇒ For other system affection:

- CBC, ECG, LFTs, KFTs, Lipid profile

Treatment

⇒ **Conservative treatment:**

Treatment of ischemia is mainly conservative:

Care of patient → stop smoking, Control diabetes, hypertension, anemia etc.

Care of foot

- avoid cutting angles of the nails, avoid tight shoes
- good hygiene & dryness of foot.

Some drugs → Trental+ baby aspirin(75mg) or clopidogrel.

⇒ **Endovascular surgery:**

A. **Percutaneous Transluminal angioplasty:**

Indications (as endarterectomy)

- Short segment affection in a large artery (2cm or less).
- Not done in occlusion below knee level.

Complications:

- Recurrence.
- Rupture of the artery.
- Distal embolization.

B. **Intraluminal stent** (after angioplasty to prevent recoil)

⇒ Surgical treatment**Indications of surgery (=late ischemia)**

- | | |
|----------------------|--|
| 1. Starting gangrene | 3. Pregangrene (CLI). |
| 2. Severe pain | 4. Tissue loss (e.g. ulcer resistant to healing) |

Surgical techniques**1. Direct arterial surgery (↑ blood supply)**

- | | |
|--------------------|------------------|
| 1. End-arterectomy | 2. By-pass graft |
|--------------------|------------------|

2. Sympathectomy**3. Amputation****Direct Arterial Surgery***Mainly in atherosclerosis with Run in & Run off***1) By-pass grafting:****Types of the grafts**

- i) Synthetic → Dacron, PTFE
- ii) Natural → Saphenous graft → (In situ, Reversed)

NB: synthetic grafts → above inguinal lig. (high flow)
 natural grafts → below inguinal lig. (slow flow)

Types of the operations**I) Anatomical By-pass****II) Extra-anatomical By-pass****Indication**

- high risk patients.
- Infected or Thrombosed prosthesis.

2) Endarterectomy:*Short segment affection in a large vessel***Sympathectomy****Indications** → cases with no run-off. → Burger's, Raynaud's disease**Contraindications**

- 1. Atherosclerosis except with tissue loss
- 2. Diabetics
- 3. Intermittent claudication.

Types

- 1. Ischemia of UL → Dorsal sympathectomy
- 2. Ischemia of LL → Lumbar sympathectomy

Disadvantage

- 1. Redistribution of blood → claudication pain
- 2. Recurrence (denervation hypersensitivity).

Amputations

Conservative amputation (Limb salvage)

- Angiography + direct arterial surgery (bypass)
- Amputation of gangrenous part only

Non-Conservative amputation

- Amputation till the level of pulsation
- Done if Limb salvage can't be done

Semi-Conservative amputation

- Angiography + direct arterial surgery (bypass)
- Amputation → below knee

Diabetic Foot Infection and Gangrene

Definition

- Term used to describe complex pathology in a diabetic patient's foot. It is related to duration and control of disease .

Predisposing factors

- 1- Peripheral neuropathy. Diminished sensations makes the patients unaware of foot injuries
- 2- Vascular affection: due to premature atherosclerosis and microangiopathy.
- 3- Compromise of the immune system. Both the humoral and cellular immunity are disturbed.

Presentation

- Neuropathic ulcer.
- Diabetic foot infection.
- Diabetic vasculopathy.
- Mixed types.
- Gangrene.

○ Neuropathic ulcer

- Patient is unaware of his foot → foot injury → neglected → ulcer (painless, deep, may reach the bone ,at pressure sites e.g heel, ball of the big toe).

Treatment :

Control diabetes, Vit B6, care of foot.

○ Diabetic foot infection:

A. Causes :

- Decrease resistance.
- glycosylation of tissue.
- +/- ischemia .
- neuropathy.

B. C/P :

- Persistent ulcer
- Severely swollen limb , red , hot & tender.
- Pus loculus , tissues becomes dark and slough .
- May spread → osteomyelitis, septic shock.

C. Treatment →**prophylactic:**

- Care of patient : control diabetes .
- Care of foot: avoid trauma, good hygiene.

Curative :

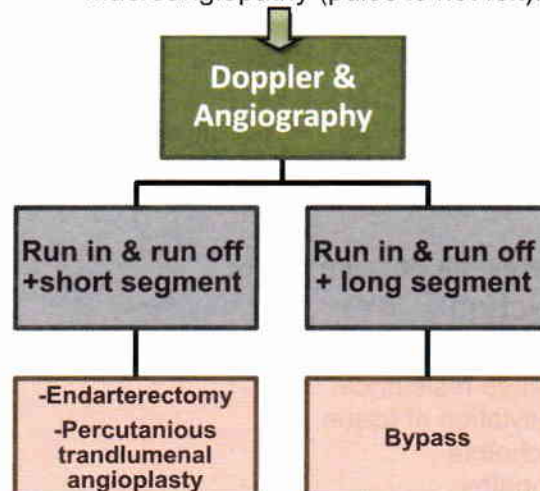
- Hospitalization.
- Rest in bed and elevation of the foot.
- Blood sugar is estimated and controlled better by crystalline insulin.
- Broad-spectrum antibiotics are started immediately before C&S. Anaerobes are commonly involved.
- Drainage of infection and debridement:
 - a. Under general anaesthesia all pockets of pus should be drained. All sloughs should be excised. A gangrenous toe needs to be amputated leaving the wound open.
 - b. If the patient is in septicemia, a major amputation is necessary to save the patient life.
 - c. Repeated dressing and repeated debridement.

○ Diabetic vasculopathy :• **Cause**

- Macroangiopathy (acceleration of atherosclerosis).
- Microangiopathy due to deposition of glycogen and lipids on the walls blood vessels.

• **C/P:** Of chronic ischemia (CB4).• **Treatment**

- Microangiopathy :(all pulses are felt, no bleeding from cut wound)
→surgical debridment , antibiotics, skin flap to promote healing .
- Macroangiopathy (pulse is not felt).

○ Gangrene

- Wet gangrene: on top of infection.
- Dry gangrene on top of chronic ischemia.
- (C/P & ttt see gangrene).

Raynaud's Syndrome

Definition

1. **Recurrent Ischemic attacks .**
2. **Precipitated by** exposure to cold or emotional stress .
3. **Commoner in UL & rare in LL.**
4. **It consists of:**
 - 1 Pallor (white) → due to VC.
 - 2 Cyanosis (Blue) → due to sluggish blood flow.
 - 3 Rubber (Red) → due to reflex VD (accumulation of metabolites).

DD

	1ry Raynaud's Disease	2ry Raynaud's phenomenon
Age & sex	young females (20 - 40 yrs)	Vary according to underlying cause
Cause	- Idiopathic . - no apparent cause(may be due to ↑sympathetic tone).	- Collagen → SLE, scleroderma. - Arterial obs. → Atherosclerosis, Burger's disease. - Nerve injury → Thoracic outlet \$, carpal tunnel \$. - Environmental → Vibrating tools - Drugs → beta blockers.
Distribution	- It affects UL (may affect LL). - It is almost always bilateral. - It is almost always symmetrical.	- May affect LL. - may be unilateral. - if bilateral it is asymmetrical.
Trophic ch. & gangrene	Absent or minimal .	More prominent.
Radial & ulnar pulse	Present .	May be absent.
Follow up	No progression & no evidence of underlying disease.	- Progressive. - manifestations of underlying dis.
Investigation	Should be directed to the suspected underlying cause	
Treatment	Conservative → Usually no ttt is needed. Care of patient - Stop smoking. - Avoid cold weather. - ttt of anemia if present. Care of Hand - Good hygiene & dryness of the hand - Hand exercise. Drugs: as chr. Ischemia + - Vasodilators → as trental. - Thyroid replacement if suspected hypothyroidism. - Baby aspirin (prophylactic for fear of vaso-obliteration).	<u>Treatment of the cause.</u> No benefit from sympathectomy as there may be : - vasculitis . - cryo-antibodies → cause VC in case of lowered bl. temp. to 36-36.5° c

Surgery → if operable

- Cervicodorsal Sympathectomy:
Immediate results are good, but may recur after one or two years (due to denervation hypersensitivity).

Thoracic Outlet Syndrome

Definition

Several anatomical abnormalities may compress the brachial plexus, the subclavian artery, or the subclavian vein near the first rib in anarrow triangle at base of neck.

Etiology

1. Cervical rib(present in 0.5% of people).
2. Scalene syndrome.
3. Costoclavicular Syndrome .
 - Compression occurs in the space between the clavicle & the first rib (congenitally narrow).
4. Malunion of fracture clavicle .
5. Pancost tumor.
6. Post fixation of the brachial plexus

Clinical Picture

- The disease is more common in females.
- Often unilateral affecting the right side due to greater use of the Rt. arm but may be bilateral.

	Neural(commonest)	Arterial(less common)	Venous(rare)
Due to	Compression of the lower trunk of the brachial plexus (C8 & T1).	Compression of subclavian or axillary artery .	Compression of the subclavian or axillary vein.
Symptoms	- Sensory → tingling & numbness along the ulnar side of hand & forearm. - Motor → Weakness & atrophy of the small muscles of the hand.	- Claudication pain induced by the use of the arm - 2ry Raynaud's phenomenon - Distal embolization may occur in the digital arteries due to embolization from post-stenotic dilatation	- Intermittent compression → venous hypertension (edema & varicosities). - Acute thrombosis (effort thrombosis).
Signs	- ↓ sensation. - ms weakness & atrophy.	- unequal pulse on both sides. - Positive Addison's Test → radial pulse becomes weak on: (1) asking patient to extend his head ,look to opposite side& takeing deep inspiration.	
- A palpable cervical rib may be felt at root of neck especially if bulbous.			

DD of Chronic Ischemia of Upper Limb

- Takayasu (aortic arch dis.)
- Burger's disease.

Investigations

1. **Plain x-ray** → cervical rib.
2. **Arteriography** → shows post - stenotic dilatation.
3. **Nerve conduction velocity** → reveal prolonged nerve conduction time.

Treatment

Mild cases with neurological symptoms

Physiotherapy & Shoulder Exercises → To develop shoulder girdle muscles.

Severe cases with vascular symptoms

Surgical Treatment → results are bad with many complications.

- Resection of the cervical rib (with its periosteum).
- Excision of the 1st rib to relieve compression .
- Division of Scalene muscle.
- Osteotomy and internal fixation of clavicle.
- Radio and chemotherapy for pancost tumor.

GANGRENE

Definition

- Death & putrefaction of macroscopic parts of tissues.

Causes

1. **Arterial obstruction** → acute or chronic.
2. **Neuropathic** → Syringomyelia, leprosy.
3. **Venous Gangrene** → Due to extensive thrombosis in peripheral veins.
4. **Traumatic** → Direct: e. g bed sores,
→ Indirect: e. g arterial injuries.
5. **Infective** → Gas gangrene.
6. **Physiochemical** → Burns, caustics, frostbite,...

Types of Gangrene

	Dry Gangrene	Moist Aseptic Gangrene
Pathogenesis (cause)	chronic ischemia → allows dryness of tissues.	- Acute ischemia - chronic ischemia with pre-existing edema (cardiac, DVT).
Pathology Putrefaction	Minimal .	Marked.
Odor	little or no odour	Very offensive
Gross	- Dry. -Mummified . - Hard . - Wrinkled. - Dark in color.	- The part remains of the same size and consistency. - Colour: at 1 st dead white the purple or greenish black..
Line of demarcation	well defined	Ill defined (no time for evaporation)

Fate	Separation → - Due to presence of line of demarcation → appears between the dead & viable tissues. - Leaving a conical stump (deeper tissue has better blood supply).	Spread → by - direct extension. - skipped lesions.
Clinical Picture	1. Lost (pulsation, Sensation, Heat, Function of affected part) fixed color changes. Press & see How Colour fades 2. The affected part is → see gross.	
	3. Minimal toxemia → better general condition.	3. Severe toxemia → poor general condition.
Treatment	See chronic ischemia - Limb salvage. - Non conservative amputation	- Amputation till the level of pulsation (acute condition). - Later on ttt of the cause if possible.

Moist Septic Gangrene

- E/O: a-Infection of a sterile gangrene (2ry infective gangrene).
b-Infection of tissues by virulent organisms (1ry infective gangrene)
- C/P:
the affected part is offensive, tense, edematous with septic pustule & there is crepitus.
- TTT.: urgent amputation.

Special Types Of Gangrene

► Decubitus Ulcer (Bed sores)

- Occurs over bony prominence (sacrum, ischial tuberosity or heels)
- Due to prolonged pressure.
- Following immobilization of paraplegic patients, elderly & diabetics.

Treatment

1. Prevention.

- Air mattress.
- Skin should be kept dry & clean.
- Frequent change of position every 2 hours.

2. Curative

- Debridement.
- Leave the wound open until healing + repeated dressing with glycerin magnesia to help separation of the slough.
- Antibiotics.
- Skin flap.

► Idiopathic Gangrene of the Scrotum (Fournier's Gangrene).

- Sudden onset of scrotal inflammation + sudden onset of gangrene.
- Due to mixed infection (synergism) by Strept., Staph., E. coli, Cl. welchii,etc.

Treatment.

- Antibiotics & wide surgical excision.
- Later on, skin graft may be needed to cover the testis.

Aneurysms

Definition

- ▶ **Pathological definition:** a sac filled with blood communicating with an artery.
- ▶ **Practical definition:** permanent localized dilatation of an artery 1.5 times than normal

Types

I. According to the etiology

1. **Congenital:**

e. g. aneurysms in circle of Willis "Berry's aneurysm" rupture causing spontaneous subarachnoid hemorrhage.

2. **Acquired** Due to :

- Atherosclerosis (commonest cause). -Trauma(→false aneurysm).
- Syphilis :
- Mycotic (i.e. arterial wall is weakened by infection) e. g. following embolization from SBE.
- Collagen disease e.g Ehlar Danlos, Behchet.

II - According to the wall of the aneurysm

1. **False:**

- Organized hematoma with fibrosed wall and compressed tissue around it.

2. **True:**

- Wall consists of all layers of the arterial wall.

III - According to the shape

1. **Saccular.**

2. **Fusiform.**

3. **Dissecting.**

Clinical Picture

Symptoms

- May be asymptomatic.
- Swelling (external) or pressure manifestation (internal).
- Ischemic manifestations due to the cause e.g. atherosclerosis

Signs

Inspection

- Swelling, along the course of an artery with expansile pulsation (systolic).

Palpation

Consistency → cystic.

Movement → across. But not along an artery.

Compression

- Totally compressible.
- Partially compressible → thrombosis.
- Proximal compression → on feeding artery → ↓ size & pulsation becomes weak.
- Distal compression → on affected artery → ↑ size & become tenser.

Auscultation → Soft systolic bruit may be heard over it.

DD

1. **Swelling over an artery** → Gives transmitted pulsation.

2. **Vascular tumors** → Heterogeneous consistency.

3. **A-V fistula** .

Complications

- Rupture → fatal Hge (suspect if diameter >6 cm, ↑elastase in blood)
- Infection.
- Pressure manifestations:
 - o Nerve → sensory, motor loss.
 - o Bone → erosion.
 - o Vein → obstruction, thrombosis.
- Ischemia due to:
 - o Thrombosis.
 - o Embolism.
 - o Associated atherosclerosis.

Investigations

1. Duplex (better or U/S ?? (سؤال عملي)).
2. CT, MRA: the best.
3. Ultrasound: as screening.
4. Arteriography: not preferred.

Treatment

1. Conservative

2. Surgery:

Indications

1. Symptomatic aneurysm.
2. Asymptomatic if the diameter is >55mm.
3. Asymptomatic in high risk patient. e.g. hypertension.

Technique

Excision → For small or unimportant artery.

Excision & grafting → For aneurysm of large artery.

Exclusion graft (open the sac, place the graft then close the sac)

Endovascular surgery: intraluminal self inflatable graft.

Abdominal Aortic Aneurysm (AAA)

Definition

CB4 + affecting abdominal aorta).

Causes

- Mainly due to atherosclerosis.
- Rarely due to other causes (mycotic, syphilitic, collagen disease).

Pathology

- Site: 95% below origin of renal arteries.
- Types: CB4.

Clinical Picture

- Silent in 75 % of cases.
- If symptomatic → pain → vague abdominal pain with backache, may be in the flanks.
- May present with complications → retroperitoneal rupture (shock, acute abdominal pain), Arterio-aortic embolism.

DD

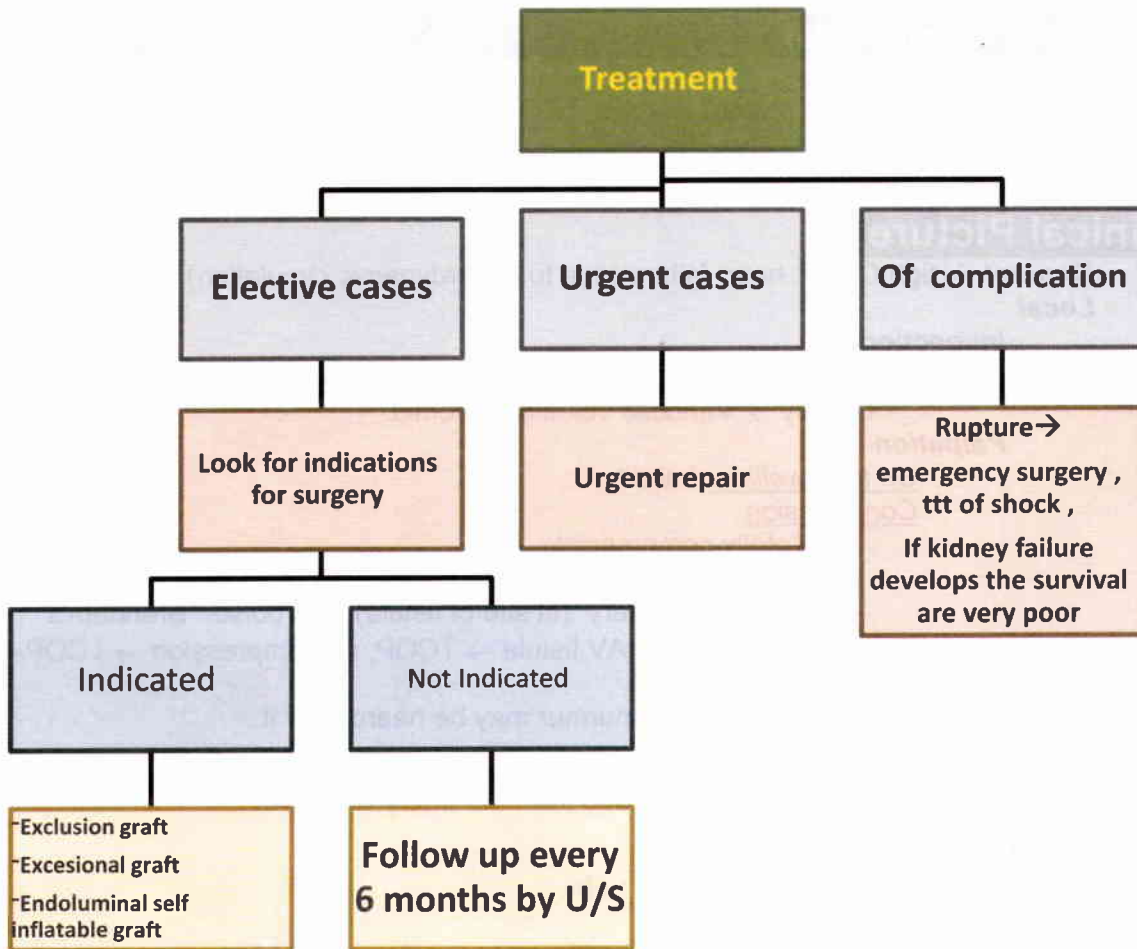
- Pseudopancreatic cyst : transmitted pulsations .

Complications

CB4

Investigations

- For aneurysm → U/S, CT , MRA, Angiography .
- For other system affection → ECG , CBC ,lipid profile .

Treatment

Arterio-Venous Fistula

Definition

- It is an abnormal shunt between an artery & vein.

Causes

- External trauma involving both artery and vein (as butcher's thigh).
- Iatrogenic trauma as catheterization.

Types

Congenital	Acquired
- May be multiple. - Affecting the small vessels (doesn't cause ischemia). - May cause local gigantism.	- Usually single. - Affecting large vessels (causes ischemia). - Common in the thigh (butcher's thigh), axilla, or neck.

Clinical Picture

General → High C.O.P. heart failure (due to hyperdynamic circulation).

Local

Inspection

- Pulsating swelling .
- Distally → Varicose veins & ischemia.

Palpation

On the swelling → thrill.

Compression

- Totally compressible .
- Partially compressible → thrombosis.
- on feeding artery (at site of fistula) → ↓pulse "Branham's bradycardia" (AV fistula → ↑COP, on compression → ↓COP → ↓pulse).

Auscultation → machinery murmur may be heard over it.

Investigations

Arteriography → shunting of the blood from the artery to the vein.

Treatment

1. Reconstruction of the artery & vein.
2. Quadriple ligation:
 - If Reconstruction is difficult (as in HF).
 - Unimportant artery.

	Deep Calf Veins	Femoral vein	Iliofemoral v.
Incidence	Commonest	Less common (usually as extension of deep calf vein thrombosis).	Rare
Pain & tenderness of	Calf muscles especially soleus.	Calf, popliteal region.	Whole L.L.
Tense tender swelling of	Foot&ankle.	calf&lower part of thigh.	Whole L.L.
Others	+ve <i>Homan's sign</i> → pain on dorsi flexion. <i>but unreliable(50% false +ve harmful).</i>		

Complications

General → pulmonary embolism.

Local

a) Early:

1. Phlegmasia alba dolens (painful white swelling d.t. partial obstruction) ± lymphoedema.
2. Phlegmasia cerulae dolens (painful blue swelling d.t. complete obstruction) ± venous gangrene.

b) Late

1. **Post phlebotic limb** (secondary VV. due to destroyed valves after recanalization of the thrombus).
2. **Venous gangrene** (plegmasia Alba & cerulae dolens).

DD

- 1- Rupture of plantaris tendon.
- 2- Calf hematoma.
- 3- Ruptured Baker's cyst.
- 4- Cellulitis: symptoms and signs of inflammation and the leg is hot and red.

Investigations

▪ To detect DVT:

1. Doppler & Duplex.
2. Recently radioactive fibrinogen → detect active uptake by thrombus).
3. Recently → spiral CT (most accurate).
4. Venography (not done now).

▪ If pulmonary embolism is suspected:

- Spiral CT
- CXR
- Ventilation /perfusion scan
- Pulmonary angiography.

▪ If recurrent DVT in young patient: Measure protein C, S. Antithrombin 3, lupus anticoagulant.

Treatment OF DVT

■ Prophylactic

- Active leg exercise, leg elevation.
- Intra & post operative pneumatic compression
- Early post operative ambulation.
- Low dose of heparin (5000 IU S.C 2 hrs before the operation then every 12 hrs till the pt. is ambulant "5-7 days")

■ Curative

General

- Absolute bed rest for 7-10 days until thrombus is adherent.
- Elevation of the limb.
- Elastic stocking.
- Vit. E & antioxidants.

Drug treatment

	1. Heparin	2. Oral anticoagulant
<u>Duration</u>	1 st 7-10 days until thrombus is adherent to vessel wall.	1 st → 3-6 ms. 2 nd → 1 yr. 3 rd → for life .
<u>Dose</u>	A loading dose of 80 IU/kg then maintenance dose of 18 IU/kg/hour by infusion pump.	Together with heparin for 2 days → then stop heparin (3 times /day → then twice then once / day).
<u>Control</u>	PTT (twice normal, N=25 to 35 sec).	PT (twice normal, N=11-14 sec.). Recently by INR.
<u>Antidote</u>	Protamine sulfate.	vitamin K.

NB. Recently:

LMW heparin → S.C 1 mg /12 h (advantages: no risk of bleeding so no need for PTT, taken twice daily only)

3. Fibrinolytic therapy

Indications:

- Very early cases → can dissolve thrombus
- Very severe cases → prevent venous gangrene

Types:

- Urokinase.
- Streptokinase .
- Recombinant tissue plasminogen activator

operative:

Intraluminal device (filter)

Types: **Greenfield filter or Miles or Spencer.**

Indications:

- 1- Contraindication for anticoagulants.
- 2- Recurrent embolism inspite of full heparinization.

Treatment of complications

- Pulmonary embolism: morphia , O₂ , thrombolytics , Anticoagulants , embolictomy.
- Post phlebotic limb: compression bandage.

Pulmonary Embolism (PE)

Cause

- Usually secondary to DVT.
- Other emboli: fat, air, infected, tumor embolism.

DD

Of Acute chest pain + shock:

Tension pneumothorax, acute Myocardial infarction, cardiac tamponade.

Clinical Picture

Fatal PE	Massive PE	Pul. infarction	Recurrent PE
Complete obstruction of the main pulm. artery → no blood returns to heart.	Obstruction of one of the 2 main branches of pulm. artery → COP will be directed to one Lung → VC → ↑ pulm. pr. → Rt. sided HF.	Small embolus → pulmonary infarction (triangle with its base towards the pleura).	Showers of small emboli → repeated infarction → pulm. Fibrosis.
Clinical picture - Acute chest pain. - Pallor. - Shock. - Sudden death.	Clinical picture - Acute chest pain. - Marked dysnea. - Marked tachycardia. - Cyanosis. - Shock → acute heart failure. - Death may occur within min. or hrs.	Clinical picture - Dyspnea (75%) due to vasospasm & bronchospasm. - Chest pain (65%). - Hemoptysis (15%). - Jaundice due to hemolysis.	Clinical picture - Tachypnea. - tachycardia, - Friction rub (rare).

Investigations

1. **Spiral CT (recent):** most accurate.
2. **Ventilation perfusion test**
3. **Blood tests**
 - ↑ LDH & serum alkaline phosphatase & normal bilirubin.
 - **Blood gases:** Hypoxia (↓PO₂ & PCO₂ due to tachypnea while in fibrosis there is ↑PCO₂).
4. **ECG** → P-pulmonale. Axis deviation to the right, right ventricular strain pattern.
5. **Chest X-ray:**
 - May be normal.
 - Wedge shaped peripheral opacity is rare.
 - ↑ or ↓ vascularity. - Pleural effusion.
6. **Pulmonary angiography**
 - Is the most accurate, yet not commonly done (invasive).
 - Can show hypoperfusion.

Treatment

Prophylaxis & TTT of DVT is very important in prevention of pulmonary embolism (mention).

☞ For pulmonary embolism :

A. Curative

Early:

- ICU: Morphine, O₂, anticoagulants, antibiotics.
- Pulmonary embolectomy.
- Thrombolytics.

Late: Heparin + oral anticoagulants.

B. In recurrent cases:

Prophylaxis against recurrence of pulmonary embolism by IVC filter.

Varicose veins

Definition

- Dilated, elongated, tortuous superficial veins of the LL.

Etiology & Types

	Primary (85%)	Secondary (15%)
E/O:	No apparent cause	Deep system is occluded
Causes (theories)	1. Congenital mesenchymal weakness 2. Cong. Valvular incompetence. <i>Ppt: Occupation with prolonged standing</i>	1. Deep vein thrombosis 2. Compression by tumor. 3. Pregnancy (commonest): 4. Arteriovenous fistula - Congenital - Acquired
Defect	in perforators & superficial system only.	in perforators, superficial & deep systems.

Pathology

Macro → Tubular, Spider, Serpentine, Saccular type (blow out or saphina varix)

Micro → thickened wall, the muscle & elastic layer are replaced by fibrous tissue

Complications

(due to ↑ venous pressure)

⇒ Venous complications:

1. **Superficial thrombophlebitis** due to sluggish circulation.
2. **Hemorrhage** after trauma (profuse) due to ↑ pressure.
3. **Edema of the L.L.** due to ↑ venous pressure.

⇒ Skin complications :

1. **Pigmentation, Dermatitis, Eczema**
2. **Varicose ulcer**: see later

3. **Malignancy:**

- On the top of varicose ulcer → Marjolin ulcer.
- Of good prognosis due to lymphatic obstruction.

4. **Talipes equinus:**

- Extremely rare occurs after long standing varicose ulcer.
- (Walking on toes relieves the pain → shortening of tendo-achillis).

Clinical Picture

	Primary	Secondary
Symptoms : cosmotic disfigurement		
Pain (venous pain)	- Mild, dull aching - relieved by elevation of the L.L. & walking.	- Marked., dull aching - Not relieved by elevation of the L.L. & walking.
Swelling (edema).	- Slight ankle swelling after prolonged standing - resolve by sleep.	- Persistent diffuse swelling - no resolve by sleep
Skin (complication)	Less common.	More common
Past history	-may be +ve F.H. of mesenchymal weakness.	- DVT
Signs :		
General	Signs of mesenchymal weakness as flat foot or kyphosis.	- Pelvic or abdominal tumors - Pregnancy
Local		
Inspection		
VV	• multiple dilated, elongated, tortuous veins • affect long or short saphenous veins • blow out at site of perforator or saphena varix.	same + 3 veins might be seen crossing the inguinal ligament
Edema	Minimal pitting ankle edema	Diffuse massive edema
Skin	Skin complication are less common	Skin complication are common
Palpation		
Thrill	on cough at Incompetent saphenopopliteal junction	
VV	Dilated, elongated, tortuous, soft, compressible, tubules	

	Primary	Secondary
Fegan sign	a defect is felt in deep fascia opposite blow out	
Percussion	Tapping test (Schwartz or chervier)	
Auscultation	machinary murmur if A-V fistula	
Sp. Tests	to detect sites of incompetent perforators and to detect the patency of deep system	

Investigations

⇒ For varicose veins :

- Doppler U/S, Duplex
- Venography → (rarely done).

⇒ For complications:

- Arteriography → In case of arteriovenous fistula.
- Plain X-ray → In case of varicose ulcer → If periostitis is suspected.
- Biopsy → If malignancy is suspected.

Treatment

1. Primary V.V:

a. Conservative treatment

Indication

- Early uncomplicated 1ry V.V.
- Patient unfit for operation.

Method

- Removal of predisposing factor.
- Elastic stocking
- Elevation of the LL.

b. Injection (Compression sclerotherapy)

Indication

- Moderate cases of 1ry V.V. with main complaint of cosmetic disfigurement.
- Residual V.V after operation.
- Lower leg perforators.
- Recurrent V.V.

c. Surgical treatment

Indications

- Large 1ry V.V.
- Presence of complication.

operations

-Trendelenburg's operation → For saphenofemoral incompetence.

-Subcutaneous stripping → The whole system is removed if there are multiple incompetent perforators.

-Triple ligation of incompetent perforator → Incompetent perforators if 2 or 3 in number (usually performed on ankle perforator).

-Recently: → *inverted stripping using pin stripper.*
→ *VNUS closure using ablation catheter.*

2. 2ry V.V. (ttt of the cause) → Only conservative.

3. Management of complications:

Pigmentation & eczema

- i) Local cleanliness & avoid itching.
- ii) Rest & elevation
- iii) Hydrocortisone & zinc oxide ointment.

Varicose ulcer

Conservative treatment For early ulcer

Rest, elevation of the limb, elastic stocking, dressing with saline.

Surgical For resistant recurrent ulcer:

- Biopsy should be taken.
- Cockett & Dodd operation.
- If failed Excise the ulcer + Cross leg skin flap

Hemorrhage → Elevation & Pr. bandage.

Marjolin's ulcer → Excision by safety margin & cross leg flap.

Talipes equines → Physiotherapy.

Chronic Leg Ulcers

➤ See D.D. Book

Venous Ulcer (varicose ulcer)

Definition

- Discontinuity of epithelium of the skin resulting in a 3D conical defect with well formed edge, margin, floor and base in relation to high venous pressure of the lower limbs.

Incidence

- Constitutes 90% of chronic leg ulcers.

Causes

- Usually post phlebitic limb due to DVT.
- Less common due to A-V fistula.
- Rarely due to primary varicose veins.

Pathology

- Recanalization of DVT is commonly followed by dysfunction of the valves of both deep veins and the perforating veins → leading to high venous pressure in the skin particularly that lying on the medial aspect of the lower third of the leg (ulcer bearing area).
- Ambulatory venous hypertension → skin capillaries become glomerulous-like → trapping of more leucocytes which become activated and release proteolytic enzymes that damage the capillaries → fibrotic process in skin and subcutaneous tissue (lipodermatosclerosis) → ulceration

Clinical Picture

👉 Clinical picture of the cause (see before)

N.B in 50% of cases there are no manifestations of varicose veins, the ulcer is due to incompetent ankle perforators only.

👉 Ulcer of the following criteria :

Site → In the ulcer bearing area (Gaitre area) just above the medial malleolus.

Number → Usually solitary.

Size

- Variable.
- In severe cases, it may encircle lower part of leg above ankle.

Floor

- Infected ulcer → dirty granulation tissue.
- Non infected ulcer → healthy granulation tissue.

Base → Indurated (hard).

Edge →

- At first → irregular, slopping & serrated then → punched out.

Margin → the skin around the ulcer is pigmented.

L.Ns → +ve if 2ry infection or malignant changes.

Complications

- Infection, Hge, osteomyelitis, periostitis, marjolin ulcer, telepius equine varius.

D.D of chronic leg ulcer see before**Investigations**

- For the cause e.g: Doppler, duplex (functional & anatomical) ,biopsy if suspecting malignancy.

Treatment**A. Treatment of ulcer*****i- Conservative treatment for early ulcer***

- Rest.
- Elevation of the limb.
- Elastic stocking → below knee, exerts 40 mmHg pressure (most important item in treatment).
- Dressing with saline & not antiseptics because of eczema. Most ulcers heal in 3-4 weeks.

B. Surgical treatment:

- If there is evidence of incompetent perforators in the leg, they can be either injected or excised.

Previous operations as Cockett's or endoscopic subfascial ligation of perforators are not commonly performed nowadays.

LYMPHATIC SYSTEM

TB Lymphadenitis

Definition

- Chronic specific inflammation of LNs caused by mycobacterium tuberculosis.

Etiology

- Causative organism:** Mycobacterium tuberculosis.
- Route:** lymphatic or blood born
- Predisposing factors:** bad general condition and low immunity.

Types

	Blood born	Lymphatic born (fibrocaceous)
Age group	More common in elderly.	More common in children.
Origin	Generalized affection in military T.B. or Blood borne from pulmonary or renal T.B.	Localized affection (from catchment area) → 1 ^{ry} complex ★ Upper deep cervical L.N. (the commonest ✓✓) → portal is tonsils Mediastinal L.N. → from pulmonary T.B. ★ Mesenteric L.N. → Intestinal T.B.
	- Tubercles are formed of epitheloid cells & giant cells (Langerhan's cells) surrounded by lymphocytes & fibroblasts. - T.B. lymphadenitis has 2 main pathological types: → Lymphatic borne & blood borne types.	
Pathology	Organisms reach L.N. through arterial supply in hilum → affection of medulla ☒ Not affecting capsule (peradenitis) ☒ No matting. ☒ No caseation. ☒ No cold abscess or sinus is ever seen clinically in this type.	Organisms reach L.N. through afferent lymphatics → affection of cortex ☒ Early affecting capsule (peradenitis) ☒ Early matting. ☒ Early caseation forming cold abscess → perforate deep fascia → Collar stud → Abscess may break down → sinuses

Clinical picture	1- Manifestations of pulmonary T.B. 2- Affected L.N. → enlarged, not tender, rubbery, not matted & discrete	C/O: → Painless swelling gradual onset, slowly progressive. O/E: 1- Manifestations of T.B. toxemia (2N2L) 2- Manifestations of Pulmonary T.B. (dyspnea, cough, expectoration & hemoptysis) 3- Affected L.N. → enlarged, not tender, firm early multiple then matted 4- Cold abscess → fluctuant (But not as hot as pyogenic abscess) 5- Multiple sinuses with undermined edge & cyanotic margins.
DD	- Hodgkin's lymphoma - Chronic non specific lymphadenitis	- L.N. secondries For Cold abscess (from other swelling in the neck) For Sinus (from sinuses in neck) thyroglossal – branchial-actinomycosis

Complications

1. Caseation & cold abscess formation
2. 2 y infection → hot cold abscess
3. Sinus & ulcer.
4. Spread.
5. Pressure on nearby structure
6. Calcification

Investigations

- 1- L.N. biopsy & culture under GA
- 2- Aspiration of cold abscess & guinea pig inoculation
- 3- Smear from sinus & examination for T.B. bacilli
- 4- For pulmonary T.B. :-
 - a) Blood → leucopenia with relative leucocytosis.
 - b) Chest x-ray → pulmonary lesions.
 - c) Tuberculin test (*good -ve test*)
 - d) Sputum examination, Zeil Nilson stain & culture on Lowenstein Jensen media.

Treatment

a) Tuberculous lymphadenitis before caseation:

- i. **Sanatorial TTT**(sun,air, good diet and nutrition).
- ii. **Antituberculous drugs:** at least Two for 9 months.
A combination of rifampicin and INH is very efficient.

- iii. **Surgical excision:** is indicated for
 - a-failure of medical TTT.
 - b-Single group of lymph nodes showing no response to medical treatment after 6 months.
 - c-Biopsy.
 - b) **Cold abscess**
 - i. **Antituberculous drugs.**
 - ii. **Drainage(+local streptomycin injection):**
 - >>**Needle Aspiration:**using Z technique to avoid sinus formation.
 - >>**Open drainage**(from non-dependent, non-pointing area):**indicated in**
 - 1. Secondary infection→acute pyogenic abscess .
 - 2. If the abscess is imminent to rupture.
 - iii. **Excision together with underlying lymph nodes is done if the nodes need excision**
 - c) **tuberculous sinus**
 - i. General antituberculous treatment
 - ii. Dressing with streptomycin powder every three days until it closes.
 - iii. Excision with underlying nodes, if resistant to conservative measures.
- TREATMENT OF BLOOD BORNE TYPE: is only medical.**

Lymphomas

	Hodgkin's	Non-Hodgkin's
Macroscopically A) L.N. - Sites: - CC's: - Cut section: B) Spleen, Liver intestine & BM	- Usually lower deep cervical → axillary → mediastinal - Enlarged + satellites, discrete early → matted later. - Pink homogenous with loss of architecture. - May be affected	- More than one group (multicentric). - Early discrete → amalgamated later.
Microscopically	Characteristic giant cell (Reed-Sternberg cell) with lymphocytes, histiocytes & PNL (pleomorphism).	
Histological types	1- Lymphocytic predominance 2- Nodular sclerosis 3- Mixed cellularity 4- Lymphocytic depletion	1-Lymphocytic lymphoma. 2- Histiocytic lymphoma. 3- Mixed cellularity. 4-Burkitt's lymphoma.

Clinically: 1-Age & sex: 2-Constitutional symptoms: 3-Swelling: 4-Pain: 5-Pressure manifest. 6-Abd Swelling:	Adolescent & middle age (bimodal) - ♂ - Fever → irregular. → Pel-Ebstien. - Itching (may be presenting \$). - Loss of wt. (> 10% in 6 m.) - Anemic manifestations. - Slowly progressive, rubbery → matted later. - specially after alcohol intake "Gordon's test" or late after infiltration. - Mediastinal L.N. → dyspnea, dysphagia, hoarseness & Horner's. - Splenomegaly, Para-aortic L.N., intestinal Obstruction & obstructive jaundice.	Extreme of age - ♂ - rapidly progressive, hard → Amalgamation later after infiltration. - As Hodgkin's - As Hodgkin's but more common.
Classification	(Ann Arbor classification) Stage I → Enlarged 1 group of LN. above or below diaphragm or single extra-nodal site. Stage II → 2 groups of LNs are affected on the same side of diaphragm or affection of 1 extra-nodal site with 1 or more groups of LNs on the same side of diaphragm. Stage III - One group of lymph node is affected above the diaphragm & another group is affected below it. - The spleen is enlarged (some considers enlarged spleen as stage IIIs). - Other consider the spleen to be a group of LNs. Stage IV → Extranodal affection especially liver & bone marrow	Lukes & Collins Classification it is based on the cells of origin, thus non Hodgkin's lymphoma is divided as - I- T-Cell type (Thymus derived: T-cell). - II- B- Cell type (bone marrow derived: B-cell). - III- Unclassifiable.

Investigations	- For diagnosis → excision biopsy if L.N. is involved. - For D.D. & staging: - CBC (pancytopenia , & ESR > 100) - LFT (↑billirubin due to obstructive, HC or hemolytic) - KFT (↑ uric acid due to tumor lysis \$) - CXR. - Abdominal U/S & C.T. - BM puncture. - Staging laparotomy is rarely used (replaced by C.T).		
Treatment	<table border="1"> <tr> <td data-bbox="483 571 1003 895"> General → Vitamins, blood transfusion, iron therapy. Definitive treatment <u>Stage I, II & IIIa</u> → radiotherapy a- If above diaphragm → Mantle. b- If below diaphragm → Inverted Y. c- If IIIa → Both mantle & inverted Y <u>Stage III b & IV</u> → Chemotherapy. MOPP (Mustine, Oncovine, Prednisone & Procarbazine) </td><td data-bbox="1003 571 1385 895"> (mainly chemotherapy as it is multicentric) As Hodgkin's lymphoma but: <u>In nodular type</u> → chlorambucil <u>In diffuse type</u> → cyclophosphamid, Vincristine & prednisone. </td></tr> </table>	General → Vitamins, blood transfusion, iron therapy. Definitive treatment <u>Stage I, II & IIIa</u> → radiotherapy a- If above diaphragm → Mantle. b- If below diaphragm → Inverted Y. c- If IIIa → Both mantle & inverted Y <u>Stage III b & IV</u> → Chemotherapy. MOPP (Mustine, Oncovine, Prednisone & Procarbazine)	(mainly chemotherapy as it is multicentric) As Hodgkin's lymphoma but: <u>In nodular type</u> → chlorambucil <u>In diffuse type</u> → cyclophosphamid, Vincristine & prednisone.
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Lymphedema

Definition

- Accumulation of fluid in extra cellular, extra vascular compartment due to lymphatic affection with edema of the overlying skin which becomes thick corrugated.

Types

1- Congenital lymphoedema:

According to age

- Lymphoedema congenita : at birth (usually aplasia)
- Lymphoedema precox : at puberty (usually hypoplasia)
- Lymphoedema tarda : in adults

2- Acquired lymphoedema:

- Filariasis (The commonest)
 - W. bancrofti --> in U.L. & scrotum
 - W. malii --> in L.L. & breast
- Repeated strept. Lymphangitis (cellulites)
- Irradiation.
- Surgical excision of LNs (e.g. after radical mastectomy)
- Malignant cells obstructing lymphatics
- Burns at site of LN.
- Injuries as circumferential scars of the limbs.

Clinical Picture

- History of the cause:
 - Endemic area for filarial
 - Past history of mastectomy or irradiation
 - Recurrent erysipelas
- Progressive edema 1st pitting LATER ON non-pitting.
- Skin then becomes thick, rough & dark (Elephantiasis)
- Ulceration with bad odor & toxemia
- Chylous ascites & hydrocele.

DD (of swollen limb)**A- Localized swelling**

- | | |
|------------------------------------|--|
| 1- Postphlebitic limb | 2- Inflammation & allergy |
| 3- AV fistula with local gigantism | 4- Adiposa delerosa (Dercum's disease) |

B- Generalized swelling: renal, cardiac hepatic ...**Investigations**

- i. Lymphangiography --> obstruction
- ii. For the cause e.g. midnight thick blood film for microfilaria.

Treatment**A. Palliative in early**

1. Rest & elevation of limb.
2. Pressure bandage.
3. Diuretics.
4. Antibiotics for infection
5. Treatment of the cause (filariasis).
 - a. Anti mosquito measures.
 - b. Diethyl carbamazine (Hetrazan)

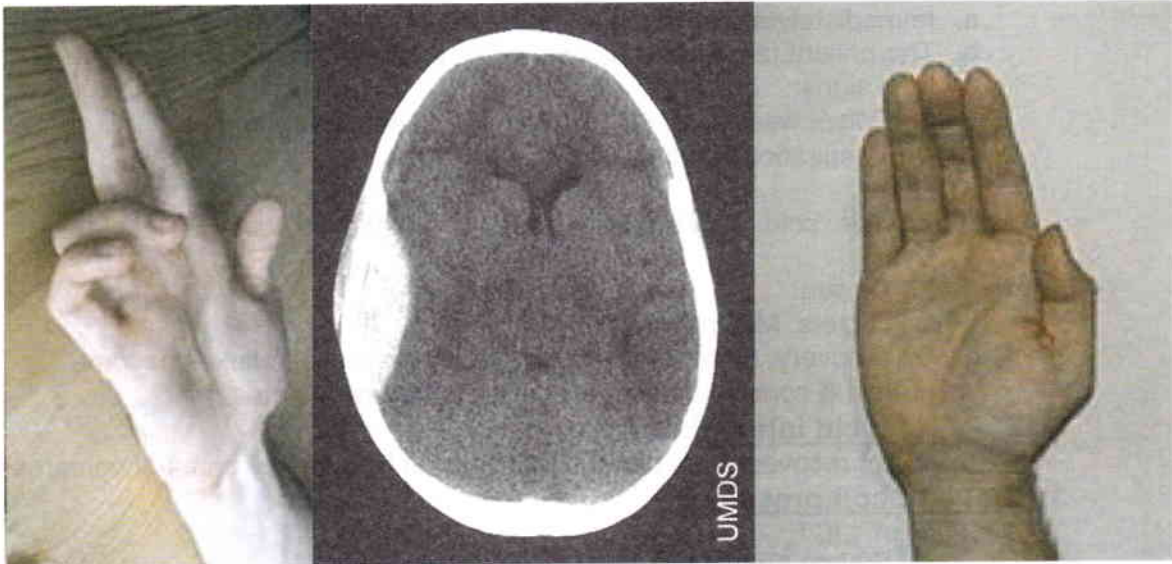
B. Surgical Treatment

Indicated IN severe disability.

1. Swiss-roll cake operation
2. Amputation: indicated if limb is hugely swollen, ulcerated & infected

5

NEURO SURGERY



Extradural Hemorrhage

Applied anatomy

Middle meningeal artery:

- External carotid A. → maxillary artery → middle meningeal A. foramen spinosum →
middle cranial fossa.

Definition

- It is hemorrhage, which occurs between skull and dura matter, commonly in temporal or parietal regions.

Etiology

- Usually due to trauma to the side of head either with or without fracture.

Pathology

- Source of bleeding:**
 - Middle meningeal artery, especially the anterior branch (most common).
 - Venous sinuses: profuse hemorrhage.
 - Diploic hemorrhage: minimal.

Clinical picture

- History → head trauma.**
- The patient passes in 3 stages:
 - 1. Stage of concussion:**
 - Immediately after trauma to the head.
 - The patient falls flaccid as one block, & loses his consciousness.
 - Vital signs:
 - Pulse: weak & rapid. ABP: ↓. Temp.: subnormal.
 - Respiration: shallow, slow.
 - Limbs:
 - Skin: cold. Muscles: relaxed. Reflexes: lost.
 - Eyes:
 - Closed. Equal, reactive pupils.
 - Sphincters: May relax.
 - On recovery, pulse, temperature, blood pressure, reflexes become normal & consciousness is regained after few minutes.
 - 2. Stage of lucid interval:**
 - Period of recovery from coma of concussion followed by coma of compression.
 - 3. Stage of compression:**
 - C/P of ↑ ICT:**
 - Severe headache.
 - Projectile vomiting.
 - Blurring of vision.
 - Papilloedema.
 - Cushing triad: hypertension, bradycardia & irregular resp.
 - Irritability.
 - Drowsiness.
 - Localizing symptoms:**
 - Coma gradually occurs with loss of reflexes.

2. Oculomotor:

⇒early:ipsilateral papillary constriction.

⇒late: ipsilateral dilated fixed pupil.

⇒The final picture before death is bilateral fixed dilated pupils.

C. Terminal Compression:

- Decerebrate rigidity d.t. compression of midbrain.

DD *Acute subdural hemorrhage.***Investigations****A- Urgent investigations:** after the patient is resuscitated & a patent airway is maintained:

- CT scan:show biconvex shadow.
- Plain X-ray: Skull Chest Cervical spine.
- Laboratory investigations: especially renal functions to assess patient's general condition.

CT scan is the investigation of choice

Complications (may occur in any head trauma)

Early	Late
1. Intracranial hemorrhage.	1. post traumatic: epilepsy, amnesia.
2. Intracranial infection.	2. Residual paralysis
3. Brain edema.	3. Hydrocephalus
4. Cerebrospinal rhinorrhea & otorrhea.	4. A-V fistula
5. Associated spinal injuries	5. Chronic subdural hematoma.

Management⇒ **First Aid** (Life-saving) ABC.⇒ **At hospital:****A- Resuscitation&1st survey:**

- I.V. line:for blood transfusion,I.V. fluids&analgesics.
- ryle tube:for feeding
- catheter:to detect urine output.

B- Continuous monitoring of:vital data,C.V.P.,urine output&level of consciousness.**C- 2nd survey** →after stabilization of pt. general condition.

I-Examination of: Scalp, skull, pupil, limb, chest, abdomen & back(For any associated injury).

*(Cervical spine injury is the most common association).*II- Glasgow coma scale

According to → Eye opening, verbal response & motor response.

III-AMPLE History:Allergy,Medications,Past medical history,Last meal&Event of trauma.

IV-Urgent investigations: as be4**D-Care of comatose patient.**I- Control ICT

- Hyperventilation is the best (m. relaxant may be needed)
 - Diuretics (mannitol or frusemide).
 - Dexamethazone.
- IV fluid intake.

II- Control fits → IV diazepam.III- Control hyperthermia→ surface cooling.IV- Care of bed sores

E-Specific treatment after localization of site of lesion by:-

- Site of trauma. - Focal signs. - Lateralizing signs.
- CT scan (the best) → Fusiform shadow between skull & brain.
- Plain x-ray (if CT is not available) → fracture & midline brain shift.

1- Craniotomy under general anaesthesia.

2- Evacuation of hematoma: by suction & irrigation by saline.

3- Control bleeder:

- Middle meningeal a. → ligation or compression by Horsley wax.
- Sinus bleeding → Gel foam.
- Dural vein → easily controlled by bone wax.

4- Management of associated conditions.

e.g. Cervical spine injury → Plaster jacket in neutral position for 3 months.

⇒ **Post operative treatment: in semisitting position****A- Observation of**

Vital data, urine output, C.V.P. & level of consciousness.

B- Drugs:

→ Antibiotics, dehydrating measures (for brain edema).

Removal of

- The drain (after 48hrs)
- The stitches after 7 days.

Acute Subdural Hemorrhage

Etiology

- Usually due to severe trauma, less commonly with cerebral laceration.

Incidence

- It is the least common of traumatic intra-cranial hematomas.

Pathology**Source**

- Usually from rupture of a large cortical vein as it crosses the subdural space.

Site

- Commonly bilateral & extensive.

Clinical Picture

- May present with picture similar to extradural haemorrhage with some differences.
- Signs can be misleading because of the extent of the underlying brain damage.

	Acute extradural hematoma	Acute subdural hematoma
Etiology	- Usually mild trauma.	- Severe trauma.
Clinical picture	- Usually mild brain damage.	- Severe brain damage & laceration.
	- Lucid interval may be present.	- Persistent loss of consciousness, no lucid interval.
	- The hematoma is usually unilateral.	- Commonly bilateral and extensive (coup & contre-coup).

Investigations	- CT → biconvex .	- C.T → cresenteric . (concavo-convex)
Treatment	- Early surgery is successful.	- The patient has serious brain damage and edema in addition to the hematoma, and so results of surgery are not very successful. Mortality rate is up to 50 %.
Prognosis	- Better prognosis	- Worse prognosis

Complications

- As extradural hemorrhage.

Investigations

- CT scan.

Management

- The same as EDH, but the dura is opened in a curciate pattern to allow rapid decompression of the brain.

Chronic Subdural Hematoma

Incidence

- More common in old age (due to brain atrophy) / alcoholics.
- Bilateral in 50% of cases.

Etiology

- Slight blow to the head, which may pass unnoticed in elderly alcoholics.

Pathology

- It results usually from rupture of the superior cerebral veins as a result of sudden displacement of the brain within the skull.
- **Cause:** Minor trauma to the front or back of the head, not enough to produce a fracture or even concussion.

Clinical picture

- The interval between the trauma and the symptoms varies between weeks to months. The symptoms are vague and consist of chronic headache, mental apathy, slowing of cerebration and the patient may even develop stupor.
- Physical signs may be absent or at most there may be unilateral or bilateral extensor plantar response. Pupillary changes are late and denote impending conization.
- The symptoms and signs wax and wane. The condition may be diagnosed as psychosis or cerebrovascular accident.

DD

- Other causes of increase intracranial tension.

Investigations

- CT is investigation of choice (hematoma will appear as hypodense area).
- Fundus examination → Papilloedema is not common.

Treatment

- Evacuation through burr holes.

Skull Injuries

Fracture Vault

1. Fissure fracture:

Cause Blunt trauma

Types

- **According to shape** → Linear, Stellate
- **According to presence of external wound** → Simple, Compound

Clinical Picture

1. **Simple fracture** → diagnosed by X-ray.
2. **Compound fracture** → seen through scalp wound

Treatment Treatment of the associated injuries.

2. Depressed fracture:

- It is localized indentation of the skull.

Causes

- Blunt trauma.

Types

1. **Simple depressed fracture**
 2. **Compound depressed fracture**
- Depressed fragment may lacerate the dura & the brain.

Treatment

1. Treatment of fracture → burr hole beside depressed segment then elevate it
2. Treatment of complication

Pond fracture

- simple depressed fracture in children
- Bone is indented rather than fractured.
- Dura is intact.
- Corrects itself by time.

Fracture Base of the Skull

Causes

1. **Trauma to the vault** → that radiates to base.
2. **Side to side compression of the head**
3. **Trauma (violence) to the base as:** → Gun shot, Blow to chin

Types

- Simple → rare.
- Compound → more common

Clinical Picture of Fracture Base

Ant. cranial fossa	Middle cranial fossa	Post. cranial fossa
Epistaxis or subconjunctival Hge	Haemorrhage from ear	Oral cavity
Escape of CSF from nose	Escape of CSF from ear	No apparent escape of CSF
Cranial nerve injuries (1,3,4,5 th) optic nerve escapes	Cranial nerve injuries 6,7,8 th cranial nerves	Cranial nerve injuries (9,10,11 th) Hypoglossal escapes

Treatment of Fracture Base

- Rest in bed**
 - Cerebrospinal rhinorrhoea → semisitting position & avoid blowing.
 - Cerebrospinal otorrhoea → lie on the opposite side & clean dry dressing
- Antibiotic.**
- Treatment of Associated brain injuries.**

Peripheral Nerve Injury

Pathology & Types

	Neuropraxia	Axonotmesis	Neurotmesis
Definition:	- Nerve concussion: physiological interruption of all its functions without any organic damage.	- interruption of the axon but the outer sheath is intact.	- Division of the nerve (partial or complete).
Effect:	-Complete motor paralysis.	-Complete motor paralysis.	- Complete motor paralysis.
Effect:	-Patchy sensory loss.	- Complete sensory loss.	- Complete sensory loss.
Regeneration:	No Wallerian degeneration and recovery occurs spontaneously within 4-6 weeks.	Wallerian degeneration in distal part for 10 days followed by regeneration 1mm/day.	- Wallerian degeneration occurs but regeneration is impossible.

Median & Ulnar nerve Injuries

	Median N. Injury	Ulnar N. Injury
Roots	C5, 6, 7, 8 & T1	C7, 8 & T1
Etiology	A. Injury at the wrist - Common due to cut wound, colle's fracture or carpal tunnel \$ B. Injury in the forearm or at the elbow - Uncommon	A. Injury at the wrist - Due to cut wound B. Injury at the elbow - Due to fractures & dislocations around the elbow or delayed ulnar neuritis(cubitus valgus deformity)
Pathology:see before		
Clinical Picture		
	A. Injury at the wrist or in the forearm	A. Injury at the wrist
	1. Motor changes:	1. Motor changes:
	Paralysis of	Paralysis of
	a) <i>Thenar Muscles</i> → Wasting of thenar eminence	a) <i>hypothenar Muscles</i> → Wasting of hypothenar eminence
	b) <i>Thenar Muscles</i> except adductor polices → Ape hand deformity	b) Medial 2 lumbricles → partial claw hand
	c) <i>Opponens Pollices</i> → Loss of apposition of the thump	c) Adductor Pollices → weak adduction of the thump → tested by froment's test
	d) <i>Weakness of abduction of the thump</i> → tested by pin touch test.	d) interossei → guttering+loss of abduction & adduction → tested by card test
	2. Sensory changes	2. Sensory changes
	Sensory loss over	Sensory loss over
	- Lateral 2/3 of palm	- Medial 1 1/2 fingers
	- Lateral 3 & 1/2 fingers	- Medial 1/3 of the palm
	- Dorsum of their terminal phalanges.	No affection of dorsal aspect of (due to sparing of dorsal cutaneous br. which passes backward 6 cm. above wrist).
	3. Trophic changes	3. Trophic changes
	→ marked due to wide area of sensory loss	→ minimal due to small area of sensory loss

	Median N. Injury	Ulnar N. Injury
	<u>B. Injury at the elbow</u> <i>As above +</i> 1. Motor changes: Paralysis of - Flexor carpi radialis → weak flexion of wrist & ulnar deviation	<u>B. Injury at the elbow</u> <i>As above +</i> 1 - Motor changes Paralysis of 1- Flexor carpi ulnaris → radial deviation of the flexed wrist.
	1- <i>Lateral 1\2 of flexor digitorum profundus → +ve Clasp test (pointing index finger)</i>	2- <i>Medial 1\2 of flexor digitorum profundus → ulnar paradox.</i>
	3- <i>a+ b → Wasting of the lateral aspect of forearm.</i>	
	4- <i>Flexors of forearm → weak hand grip & weak flexion</i>	
	2. Sensory changes <i>As before in the hand.</i>	2. Sensory changes - <i>As before in the hand</i> + <i>dorsal aspect is affected</i>
Invest.	<u>Quinizarine powder test</u> → loss of sweating from affected area Galvanic stimulation > faradic.	
Prognosis	Bad since it is a mixed nerve controlling fine muscles	
TTT	a- <u>Closed injury:-</u> - Expectant TTT for 6 m. o Electric stimulation of m. o Physiotherapy. o Splint in position of best function. If no response:- - Exploration of n. - Remove any neuroma, glioma or intervening T. - Trim & anastomosis the ends. If wide gap:- - N. graft or n. transposition (infront of med. Epicondyle) b- <u>Open injury:-</u> - Only debridement (no 1 ^{ry} repair) - Repair If not infected → delayed 1 ^{ry} repair (after 3 w.) If infected → 2 ^{ry} repair (after 6 w.)	

DD (of claw hand):

1. Klumpke's palsy & costo-clavicular \$.
2. Combined ulnar & median n. injury.
3. Volkmann's ischemic contracture.
4. Dupuytren's contracture.
5. Post-burn scar contracture.

Radial Nerve Injury

Roots of Origin

- C5, 6, 7, 8 & T1

Etiology

- A. In the axilla → fracture of upper end of humerus & pressure by crutches
- B. In the spiral groove → Fracture middle 1/3 of humerus & Saturday night paralysis
- C. Injury of the posterior interosseous n. → Fracture or dislocation around the elbow.

Pathology See before

Clinical Picture

A. Injury in the axilla:

1. Motor changes → *Paralysis of the following muscles:*

- a) Triceps → the elbow (flexion)
However can be extended only by its own weight.
- b) Supinator → pronation of forearm
However can still be supinated by the biceps.
- c) Extensors of wrist → **wrist drop**
- d) Extensors of fingers & thumb → **fingers drop**
However fingers can be extended by the lumbricalis & interossei if the wrist & fingers are supported

2. Sensory changes → Sensory loss limited to a small area at base of thumb

B. Injury in the spiral groove → wrist drop + finger drop.

C. Injury of the posterior interosseous n.: → Only finger drop

Investigations

- Quinizarine powder test → loss of sweating from affected area
- Galvanic stimulation > faradic.

Prognosis

- Good (purely motor nerve controlling coarse movements).

Treatment

A- Closed injury:-

- Expectant TTT for 6 m.
 - Electric stimulation of m.
 - Splintage in position of best function.
(The hand is hyperextended in a cock up splint)
- If no response:-
 - Exploration of n.
 - Remove any neuroma, glioma or intervening T.
 - Trim & anastomosis the ends.
- If wide gap: N. graft or n. transposition

B- Open injury:-

- Only debridement (no 1st repair), just mark ends by prolene suture.
- Repair
 - If not infected → delayed 1st repair (after 3 w.)
 - If infected → 2nd repair (after 6 w.)

Brachial Plexus Injuries

Etiology

Open injuries

- Gun shot or stab wound

Closed injuries

- Fracture of the clavicle.
- Hyperabduction of the arm.
- Birth traction injury.

Pathology

- See general

Clinical Picture

Complete brachial plexus injuries: damage of all roots(C5,6,7,8&T1).

- Motor changes: paralysis of all muscles of the upper limb except trapezius
- Sensory changes : Anesthesia of whole upper limb except:
- Medial side of arm (supplied by intercosto-brachial nerve (T2).
- Skin over upper part of deltoid muscle (supplied by supraclavicular nerve
- Horner's syndrome: due to sympathetic paralysis.

Upper trunk injury: (C5 - C 6) Erb'duchenne paralysis

- Motor changes: policeman's tip deformity.(waiter's tip position)
- Sensory changes: Anesthesia over the deltoid muscle(absent if the fifth nerve is involved only).

Lower trunk injury: (C8 - T1) Klumpke's paralysis.

- Motor changes: complete claw hand.
- Sensory changes: anesthesia along the medial aspect of the forearm & the medial 1&1/2 fingers.
- Horner's syndrome: due to sympathetic paralysis.(it indicates bad prognosis)



Carpal Tunnel Syndrome

Definition

- Compression of median nerve inside carpal tunnel under flexor retinaculum

Types

- **Acute:** due to fracture of the carpal bones.
- **Chronic:**
 - Primary - Due to thickening of flexor retinaculum. - common in ♀
 - Secondary → Due to osteoma of carpal bones.

Clinical Picture

- Of chronic primary carpal tunnel syndrome.

Symptoms

Early → Pain in the hand along distribution of median nerve.

Late → Weakness, parathesia & numbness.

Signs (as median nerve injury at wrist except sensory loss at palm)

Early → tenderness on percussion on median nerve

Late → atrophy of thinner muscles & sensory loss along digital branches of lateral 3 ½ fingers.

Treatment

Division of the flexor retinaculum.

Fracture Spine

Incidence

- Most commonly affected spine are the junction of mobile and rigid parts at lower cervical and dorso-lumbar regions.

Etiology

1. Trauma.

- Hyper-flexion trauma. This is the most frequent trauma, e.g., fall of a heavy object on the back of the trunk or head.
- Hyper-flexion and rotation.
- Vertical compression trauma:
 - fall from height on the feet.
 - Fall on the head.
- Hyper-extension injuries are uncommon.

2. pathological fractures:

- Metastases.
- Osteoporosis.

Types

► According to morphology:

- Wedge compression fracture
- Fracture dislocation.
- Dislocation.
- Comminuted (burst) fracture.
- Avulsion fractures of transverse and spinous processes are not accompanied by neurological injury and require no special treatment.
- Dislocations and fracture dislocations of the atlas and axis vertebrae are serious as they may be fatal because of transection of the cord above the level of innervation of respiratory muscles.

► According to stability:

Stability of the spine fractures depends on the integrity of the posterior ligaments; the interspinous, supraspinous and ligamentum flavum

- Stable fractures. Have intact posterior ligaments, e.g., wedge compression, comminuted and avulsion fracture of the transverse and spinous processes.
- Unstable fractures. Have torn posterior ligaments and are liable to injure the cord and nerve roots, these include fracture dislocation and pure dislocation.

Clinical Picture

- 1- Spine injury is suspected after major trauma or if the patient is unconscious after the injury.
- 2- Local signs
 - Slight kyphosis may be felt or visible.
 - There is local tenderness over the spinous processes. No gap is felt between the spinous processes in a stable fracture and vice versa in unstable fractures.
 - No attempt should be made to test for back movements as this may induce paraplegia.
- 3- Neurological assessment
 - Motor power.
 - Sensations.
 - Reflexes.

Complications

- 1- Spinal cord and/or root injury including roots of cauda spina
- 2- Shock may be:
 - a- Spinal shock
 - Sympathetic affection leading to loss of vascular tone & bradycardia
 - loss of muscle tone → venous pooling & hypovolemia
 - b- Hemorrhage lead to true hypovolemia

Investigations

- A- **For diagnosis:** plain X-ray spine, AP and lateral
- B- **For complications:** CT and MRI
- C- **For detection of cause** of pathological fracture

Treatment

A. Initial management

- ▣ When fracture of the spine is suspected movements of the spine must be prevented.
- ▣ A cervical collar is applied and the head is immobilized during transport and during examination.

B. Stable fractures

1. **Wedge compression fracture.** The patient is put in bed and is taught extension exercises for the back muscles
2. **Comminuted fracture.**
 - ▣ A plaster jacket is applied in the neutral position(straight spine;not hyperextended) for 3 months.
3. **Avulsion fractures** of the transverse or spinous processes.Rest and analgesics.

C. Unstable fractures

1. **No neurological deficit**
2. **Cervical spine:**
 - ▣ Traction using tongs for 6 weeks is followed by a cervical collar for one month.Open reduction and internal fixation by plates may be needed.
3. **Thoraco-dorsal spine:**
 - ▣ Reduction by gentle extension is followed by fixation in a plaster cast in mild extension. If closed reduction is not possible, open reduction and internal fixation is the alternative.
4. **Evident cord transaction:**
5. **Care of paraplegic patient.**

6

UROSURGERY



Urinary Stones

Incidence

- More in middle-aged males.
- Common in Egypt due to bilharziasis and hot climates.

Predisposing Factors

1. Metabolic errors (1ry stone)

a- **Calcium stones:** (mainly Ca oxalate and Ca phosphate):

⇒ *Associated with:*

- Hyperparathyroidism (commonest cause).
- Vitamin D intoxication.
- Bone metastasis.
- Immobilization.
- Sarcoidosis.
- idiopathic Hypercalcuria.

b- **Uric acid stones:** gout.

c- **Cystine stones:** in cystinuria

2. Infection (2ry stone)

- Infection leads to stone formation (usually phosphate stones) through:
 - Changing pH of the urine → alkaline urine.
 - Providing a nidus (pus cells + epithelial debris) for stone formation.

3. Others

a- **Diet:**

- Milk and its products → calcium stones.
- Mango, tomatoes, berries, spinach → oxalate stones.
- Liver, kidneys, ducks → urate stones.
- Eggs, meat, fish (high sulphur containing proteins) → cystine stones

b- **Hot climate:** hot weather → sweating → concentrated urine → stone.

c- **Primary renal disease:**

e.g. congenital anomalies of urinary tract predispose to stasis, infection & stone formation.

Pathology (Types of stones)

Types	Oxalate	Cystine	Phosphate	Urate
Surface	Spiky	Smooth	Smooth	Smooth
Consistency	Hard	Soft	Hard	Hard
1 ry, 2 ry	1ry	1 ry	2 ry	1 ry
Radiological	Opaque	Opaque	Opaque	Lucent

Clinical Picture

Type of patient

- Middle-aged male.

Symptoms

1. **May be asymptomatic:** (accidentally discovered)

2. **Pain:** α site of stone:

- ⇒ Renal stones: dull aching pain in the flanks due to stretch of renal capsule.
- ⇒ Ureteric stones: (ureteric colic)
 - Severe **agonizing pain, loin to groin** (and to the tip of penis if at the intramural ureter).

- Sudden onset, **rarely > 8 hours**.
- Associated with **nausea, vomiting** and if intramural → strangury (painful passage of few drops of urine)
- Hematuria may occur after attack of ureteric colic.

⇒ Bladder stones:

- Varies from slight discomfort to severe pain in the suprapubic region referred to the tip of penis or vulva at the end of micturation (due to contraction of the bladder around the stone)
- More during daytime and aggravated by movement and relieved by lying down.
- Associated with:
 - **Difficulty in micturation:** strangury with few drops of blood stained urine and sense of incomplete evacuation.
 - **Frequency:** first diurnal then diurnal and nocturnal (after infection)

⇒ Urethral stones:

- Migratory stone: history of renal colic 2-3 days before.
- During the last micturation → sudden arrest of urine.
- Followed by acute retention.

Acute colic is reflexly associated with nausea and vomiting.

3. Symptoms of complications:

e.g. painful hematuria, pyuria, anuria or retention according to the site.

Signs

1. General: (complications)

- ⇒ Manifestations of infection: fever, tachycardia.
- ⇒ Features of uremia: → late, if both kidneys are damaged.

2. Local (abdominal):

- May show a **tender renal swelling** (hydronephrosis or pyonephrosis).
- **Suprapubic tenderness** (stone bladder).
- Huge stone in the bladder might be felt by bimanual examination.
- Stone in the penile urethra → felt on the under surface of the penis.

- In ureteric stone: rigidity of lateral abdominal muscles and not rectus abdominis.

Complications (HIMOM)

Hematuria (painful)

Due to ulceration:

- Total** → in renal, pelvic or ureteric stones.
- Terminal** → in bladder stones.
- Initial** → in prostatic urethral stone.
- Not related to micturation** → in distal urethral causes.

Infection

- **Pyelonephritis** → loin pain, fever, rigors, frequency and dysuria.
- **Pyonephrosis** → painful loin swelling with marked constitutional symptoms.
- **Cystitis** → suprapubic pain with day and night frequency & dysuria.

Migration

- Leading to change in the character of pain according to the site.

Obstruction⇒ **Back pressure:****A- Above the urinary bladder:**

1. Partial → ipsilateral hydroureter and hydronephrosis:
 - Bilateral cases → renal atrophy & renal failure.
2. Complete → calculus anuria:
 - If bilateral ureteric obstruction or unilateral in the only functioning kidney.

B- At bladder neck or urethra → acute retention

- Painful desire with full bladder

Malignancy

- Squamous cell carcinoma on top of leukoplakia.

DD*Causes of loin to groin pain:*

1. **Renal calculi:**
 - Kidney.
 - Ureters.
2. **Infection:**
 - Pyelonephritis.
 - Cystitis with ascending infection.
3. **Hydronephrosis.**
4. **Renal cell carcinoma.**
5. **Carbuncle of the kidney.**

Investigations**A- For diagnosis**1. **Laboratory:**⇒ **Urine examination:**

- RBCs in 90% of cases.
- Pus cells.
- Crystals:
 - a. Envelope-like → oxalate stone.
 - b. Hexagonal plate → cystine stone.

2. **Radiology:**1. **Plain x-ray (KUB film):** (shows 90% of urinary stones).2. **U/S:**

- Show both radio-opaque & radiolucent stones.
- Renal parenchymal thickness.
- Not reliable in visualization of uretric calculi.

3. **IVU: (intravenous urography = descending urography):**

- Done if KFTs are within normal.
- Anatomical values:
 - To detect radio-lucent stones (10%) → filling defect.
 - To show any back pressure.
- Functional values: differential kidney function.
- Contraindication:
 - Blood urea > 50 mg% - During attacks of pain.

4. **Ascending urography (retrograde urography):** if renal functions are impaired.5. **Isotope scanning of the kidney (rarely needed):**

- Static (DSMA).
- Dynamic (DTPA).

3. **Instrumental: (Cystoscopy)**

- May show bladder stones.
- May show bloody efflux from the ureteric orifice on the affected side or an impacted stone.

B- For the cause (mainly in recurrent and bilateral cases)

1. Serum calcium, phosphorus & parathormone → for hyperparathyroidism
2. Stone analysis: if the stone is retrieved.
3. Uric acid in blood.
4. Calcium, uric acid, cystine in 24hours urine.

C- For complications

1. Urine analysis: RBCs, pus cells, culture & sensitivity
2. KFTs: BUN and creatinine.

Treatment**I- Acute attack**

1. Hospitalization (if needed).
- 2- Potent analgesics (morphine)
3. Prophylactic antibiotics.

II- Definitive treatment

(After resolution of the acute attack or in accidentally discovered stones)

1. **Conservative management:**

(If small < 1 cm, with no complications)

- Ample of fluids, Antispasmodics, Acidification of the urine, Antibiotics & follow up.

2. **Active management (according to site & size):**

Instrumental (if >1 cm or complications) or surgical (if failed or unavailable instrumental)

➤ **Renal stone:**

- If 1-2 cm → Extra-corporeal Shock Wave Lithotripsy (ESWL)
- If infected, impacted or failure of ESWL → percutaneous nephro-lithotomy.
- If > 2 cm or distal obstruction → Surgery:
 - Renal stone → Nephro-lithotomy.
 - Pelvic stone → Pyelo-lithotomy.
 - Stag horn stone → extended pyelo-nephro-lithotomy.
- Partial nephrectomy → if multiple stones causing cavitations in the lower calyx.
- Total nephrectomy → if the kidney is non-functioning provided that the other kidney is normal.

Renal or pelvic stone > 2 cm can be reduced by PCNL then ESWL

➤ **Ureteric stone:**

- Retrograde endoscope with laser.
- ESWL in situ if detected by U/S.
- If upper or middle ureter → push bang technique then ESWL.
- If within 5-6 cm from ureteric orifice → endoscopic removal by dormia basket.
- If at urteric meatus → cystoscopic ureteric meatotomy by diathermy + stone extraction.
- If failed → uretrolithotomy.

➤ **Bladder stone:**

- If 1-2 cm. → Cystoscopic litholapaxy → if failed or contraindicated → percutaneous suprapubic litholapaxy
- If > 2 cm. → Suprapubic cystolithotomy.

➤ **Urethral stone:**

- Prostatic urethra:
 - Push to bladder to be crushed by litholapaxy & evacuate fragments.
 - If failed → Suprapubic cysto-lithotomy.
- Penile urethra:
 - Local anesthetic gel & try forceps extraction by urethroscope
 - If impacted → Urethrotomy + suprapubic cystostomy.

3. **TTT of complications:**

➤ **Hydronephrosis:**

- If the kidneys are still functioning → removal of the stone.
- If the kidneys are not functioning:
 - If unilateral & the other kidney is functioning → nephrectomy.
 - If bilateral → nephrostomy tube, then treatment of the better functioning kidney; if still not functioning → dialysis then transplantation.

➤ **Pyonephrosis:** as before but add parenteral antibiotics.

➤ **Acute retention of urine:** (Relief of retention)

- Catheterization or suprapubic cystostomy to be prepared for definitive TTT.

➤ **Calculus anuria:**

- Relieve by ureteric catheter with trial of endoscopic extraction.
- If failed → Nephrostomy tube then uretero-lithotomy.

4. **Special problems:**

➤ **Recurrence:** avoided by:

- Medical treatment: ample amount of fluids, antibiotics.
- Treatment of the cause: as hyperparathyroidism.

➤ **Bilateral stones:** start by the better functioning kidney then treat the other kidney after 2-3 months with exception:

1. Painful side first if pain persists.
2. Pyonephrosis first to save life.

➤ **Multiple stones:** start by urethra, ureter, kidney then bladder

Prevention of urinary stones recurrence

▶ **General measures:**

1. Excess water intake.
2. The stone should be chemically analyzed.
3. Restriction of diet containing high crystals according to chemical composition of the stone.
4. Treating infection and other causes of stone formation.
5. Follow up of stone formers to detect early recurrence.
6. Metabolic work-up to know etiology of the stone.
 - Serum Ca and phosphorus to exclude hyperparathyroidism.
 - 24-hour urine collection for the following whose normal values are:
 - Ca <300 mg.
 - Uric acid <800 mg.
 - Oxalates <40 mg.
 - Citrates 300-900 mg.

▣ CALCIUM OXALATE STONES:

1. Avoid diet rich in oxalates.
2. Hydrochlorothiazide 50 mg bid aids in the dissolution of Ca oxalate stones.
3. Citrates 5 mg bid inhibits crystallization of oxalates.

▣ URIC ACID STONES:

1. Avoid diet rich in purines.
2. Rule out myeloproliferative or neoplastic diseases.
3. Urine should be kept alkaline, e.g. by NaHCO_3 1 gm tds.
4. Allopurinol 30 mg/day is indicated in patients with hyperuricemia.

▣ AMMONIUM MAGNESIUM PHOSPHATE (STRUVITE):

1. Aluminum hydroxide orally restricts phosphate absorption.
2. Long term antibiotics to eradicate UTI.
3. Avoid indwelling catheters.
4. Increase urine acidity by oral administration of 1 gm vitamin C daily.

Calculus anuria

Definitions

- **Anuria** → no urine excretion for 12 hours.
- **Oliguria** → urine excretion less than 400 ml in 24 hours.

Etiology

- **Bilateral complete ureteric obstruction by stones.**
- **Unilateral complete ureteric obstruction by stone if the other kidney is:**
 - Congenitally absent.
 - Pathologically destroyed.
 - Surgically removed.

Clinical picture

- **There may be history of urinary stones.**
- **Stage of onset:**
 - Sudden onset of severe ureteric colic (describe)
 - Enlarged kidney on the affected side.
 - No urine, no desire, empty bladder.
- **Stage of tolerance (3-8 days):**
 - Pain gradually disappears.
 - Blood urea progressively rises.
- **Stage of uremia:** After few days, uremia is established.

DD

	Calculus anuria	Acute retention of urine
History	ureteric colic	severe supra pubic pain
Examination	bladder is empty	bladder is distended
Catheterization	catheter is passed → no urine	catheter is passed → brings urine

Investigations

For diagnosis

1. **Plain x-ray:** may detect radio-opaque stone.
2. **Ultrasonography:**
 - ⇒ Is very important.
 - ⇒ It reveals a distended pelvi-calyceal system on the affected side. It should be remembered that a large hydronephrotic kidney is usually the not the side of acute obstruction as it is often non-functioning.
3. **Ascending pyelography:** diagnostic and therapeutic (see below)
4. **Cystoscopy:**
 - Impacted stone at the ureteric orifice. - Helps to pass ureteric catheters.
5. **Urethral catheter:** to exclude retention.

For complications

- Blood urea & electrolytes

IVU is contraindicated in anuria

Treatment

Placement of bilateral ureteric catheters:

- 1- If obstruction relieved → antibiotics and antispasmodics for 48 hours, then remove the ureteric catheters:
 - If the stone passed spontaneously → measures to avoid recurrence (e.g. Ample amounts of fluids, Antibiotics)
 - If the stone is impacted → ureteroscopy with removal of stone:
 - If removed → measures to avoid recurrence.
 - If failed → surgical removal of the stone (ureterolithotomy)
- 2- If obstruction is not relieved → nephrostomy, then removal of the stone surgically (ureterolithotomy) later on.

Retention of Urine

Definition

- Failure of bladder evacuation with functioning kidneys.

Types

1. **Acute retention:**
 - Sudden complete failure to pass urine inspite of desire, **Painful**.
2. **Chronic retention:**
 - The patient can pass urine but some urine always remains in the bladder (residual urine increases), **Painless**.
3. **Retention with overflow (false incontinence):**
 - Micturation is replaced by a continuous dribbling of urine from an overdistended bladder on top of chronic retention, **Painless**.

Acute Retention of Urine

Etiology

1. **Mechanical lower urinary tract obstruction:**
 - **Urethra:** Rupture, stones, blood clot.
 - **Prostate:** SEP, acute prostatitis, cancer.
 - **Bladder:** stone, cancer.
 - **Pelvic mass:** Incarcerated gravid uterus.
2. **Paralytic:**
 - Acute stage of spinal cord injury.
 - Surgical denervation of bladder.
 - Drug induced: e.g. tranquilizers or propanthine.
3. **Hysterical:**
4. **Reflex:** postoperative, especially after anorectal operations

The most common causes are:

- Senile enlargement of the prostate
- Stone (in urethra)
- Trauma
- Postoperative

Clinical Picture

Symptoms

1. **Symptoms of acute retention:**
 - Suprapubic cramp like **pain**.
 - **Sudden inability** to pass urine in spite of desire.
2. **Symptoms of the cause:**
 - 1- **Stone:**
 - UB → supra-pubic pain referred to the tip of penis & terminal hematuria.
 - Urethra → urethral pain referred to the tip of penis & initial hematuria.
 - 2- **SEP** → prostatism (LUTs):
 - (Dysuria, night frequency, hesitancy, weak stream, post-micturation dribbling, double micturation & attack of retention)
 - 3- **History of trauma or post-operative.**

Signs

⇒ Local:

- **Urinary bladder:**
 - Inspection: suprapubic bulge
 - Palpation: suprapubic tender pyriform swelling
 - Percussion: suprapubic dullness.
- **Kidney:** may be loin swelling due to back pressure.
- **Urethra:** palpate the urethra for stone
- **DRE:** may show the cause e.g.
 - BPH → soft, symmetrical, smooth, preserved sulcus, mobile rectal mucosa.
 - Prostatic carcinoma → hard, asymmetrical, nodular, lost sulci, fixed rectal mucosa.

DD

- Acute retention from calculi anuria → (see calculus anuria).

Investigations

For diagnosis

- Diagnosed clinically.
- Pelvic U/S → shows full bladder.

For the cause

- X-ray → stone, or carcinoma.
- Transrectal U/S → SEP, cancer prostate

Management

Treatment of retention

A. Reflex retention → never rush to catheter.

You are a doctor not a plumber → don't be catheter minded

⇒ Conservative:

1. Strong analgesics for pain.
2. Allow the patient to get out of bed.
3. Warm applications on the lower abdomen & perineum.
4. Running tap in front of the patient.
5. If the above measures failed → catheterization.

B. Mechanical obstruction

- Catheterization using *Nelaton's* catheter (removed after drainage of urine)
- **If recurrent retention** → Indwelling Foley's catheter & prepare patient for surgery.
- **If failed** → Suprapubic catheter (Cystofix ®) under local anesthesia

Treatment of the cause

1- Urethral stone:

- a- Prostatic urethral stone :
 - **Push** to bladder to be crushed by litholapaxy & evacuate fragments.
 - **If failed** → suprapubic cysto-lithotomy.
- b- Penile urethral stone: **local anesthetic gel & try forceps extraction.**

2- Bladder stone:

- **If < 2 cm** → cystoscopic litholapaxy. - **If > 2 cm** → cysto-lithotomy.

3- BPH

- a- **Medical** treatment may be used if it's the 1st attack of retention.
 - 5 alpha reductase inhibitor (Proscar ®) - α-blockers.
- b- **Surgical:-**
 - Trans-Urethral Resection of Prostate (TURP).
 - Open surgery (transvesical or retro-pubic prostatectomy)

Chronic Retention of Urine

- Gradual bladder distension with residual urine with no pain making the patient unaware leading to retention with overflow.

Causes

- **Urethra:** stricture. - **Prostate:** BPH, carcinoma. - **Bladder:** BNO, carcinoma.
- **Pelvic mass:** fibroid, ovarian or cervical tumors.

Symptoms

- Suprapubic discomfort. - Painless urinary bladder swelling.
- First, frequency especially nocturnal then later on bed wetting.
- Symptoms of the cause (BPH) → see before.
- Symptoms of complications (hydronephrosis) → loin pain + swelling.

Signs

- Bladder is **full** (may reach to the level of umbilicus) & **not tender**.
- Signs of the cause (BPH → DRE → soft, symmetrical, smooth, preserved sulci, mobile rectal mucosa)
- Signs of complications (hydronephrosis → tender loin mass).

Investigations

For diagnosis

- Like acute retention + urine analysis.

For the cause

- e.g. transrectal U/S → BPH.

For complications

- IVU to assess back pressure.

Treatment

Treatment of chronic retention

- Foley's catheter or condom catheter.

If urea >100 mg% → evacuate bladder gradually
(to avoid reactive hyperemia of renal tissue which may cause haematuria or renal shutdown)

Treatment of the cause

- BPH (medical or surgical).
- Carcinomas → according to its stage.
- Urethral stricture → dilatation, internal urethrotomy or urethroplasty.

Treatment of complications

- Hydronephrosis.

Hydronephrosis

Definition

- Aseptic** distention of the pelvi-calyceal system due to chronic **partial obstruction**.

Causes

		Congenital	Acquired
Unilateral	Renal	Horse-shoe kidney, aberrant renal vessels crossing the pelvis, PUJ. Obstruction	Stones, tumors, renal TB
	Ureteric	Ureteric reflux, ureterocele, stenosis	Stones, tumors, TB, pressure from outside (as cancer cervix), retrocaval ureter
Bilateral	Urinary bladder	Congenital contracture of the bladder neck	Stones, BNO, tumors, T.B
	Prostatic		Stones, BPH, cancer prostate
	Urethral	Post. urethral valve, phimosis	Stones, urethral stricture

*The commonest cause of bilateral hydronephrosis is S.E.P.
The commonest cause of unilateral hydronephrosis is stone.*

Pathology

- a) **Pelvic type:** (in extra-renal pelvis)
 - The kidney is enlarged.
 - First, pelvis is dilated then calyces and late the kidney is formed of loculi **communicating with the renal pelvis.**
- b) **Renal type:** (in intra-renal pelvis)
 - Destruction of renal parenchyma occurs more rapidly.

Clinical Picture

C/P of hydronephrosis

Symptoms:

- Long standing dull aching pain & heaviness in loin (stretch of the renal capsule).
- Pain becomes worse on drinking large amount of fluids.

Signs: mass in renal angle, may be slightly tender.

C/P of the cause

- Stone → colic, painful hematuria. - BPH → prostatism (LUTs).
- TB → constitutional symptoms, frequency.

C/P of the complications

- e.g. Hypertension, fever (in infections) and in late cases → renal failure.

Complications

- ♦ **General:** renal hypertension, renal failure.
- ♦ **Local : As any cyst**
 - Infection → pyonephrosis. - Pressure atrophy.
 - Others: calcification – rarely rupture or Hemorrhage.

DD

of renal mass

(Polycystic kidney, hypernephroma, liver swellings on the right side, splenomegaly on the left side)

Investigations

For diagnosis

- **U/S:** detects the **size** of the kidney and the **thickness** of the renal cortex.
- **IVU:**
 - Dilatation of renal pelvis. → distortion of PUJ.
 - Clubbing or Ballooning of the calyces, widening of the waist.
- **Ascending pyelography:** if IVU is contraindicated due to renal failure .
- **Renal isotopic scan:** to show the remaining functioning parenchyma.
- **Plain X-ray:**
 - obliterated psoas shadow. → Stone, calcifications.
- **Urine analysis:** polyuria of low specific gravity

For complications

- **KFTs:** for renal failure
- **CBC & ESR:** to exclude infection.

For the cause

- **Transrectal U/S** → for SEP. - **Cystoscopy** → bladder lesions (bilharziasis or tumor).

Treatment

- 1- **Unilateral** → if functioning (20% of total renal function) : ttt of the cause.
→ non-functioning : nephrectomy provided the other kidney is normal.
- 2- **Bilateral** → if functioning : ttt of cause.
→ if non-functioning : nephrostomy then:
 - If kidney functions improved → treat the better functioning kidney.
 - If no improvement → renal transplantation.

Treatment of the cause

- 1- **Congenital:** (treat in-utero if severe)
 - a- Pelvi-ureteric lesions → Anderson Hynes principle.
 - b- Ureterocele → diathermic fulguration.
 - c- Narrow external meatus → meatotomy.
 - d- Phimosis → circumcision.
- 2- **Acquired**
 - a. Stone → endoscopic or surgical removal.
 - b. SEP → Trans-urethral resection of prostate (if small).
→ Open prostatectomy (if large).

Treatment of complications

- Renal hypertension: ACE inhibitors, nephrectomy of the affected kidney provided that the other kidney is normal.
- Renal failure → replacement therapy (dialysis or transplantation)
- Pyonephrosis → drainage & parenteral antibiotics.

➤ Acute obstructive conditions in urinary tract:

1. **Acute retention of urine.**
2. **Calcular anuria.**
3. **Ligation of both ureters during surgical procedures.**

Renal Injuries

Etiology

1. Blunt injuries {the commonest (80-85%)}: it may be
 - Direct: e.g. car accidents.
 - Indirect (less common): e.g., falling from a height or fracture rib.
2. Penetrating wounds e.g. knives or bullets.
3. Iatrogenic injuries occur during surgery.

Pathology

Renal injury may be one of the following degrees:

1. Bruising and ecchymosis are the most frequent.
2. **Superficial tear:**
 - Affects the cortex.
 - This leads to subcapsular hematoma or perinephric hematoma.

3. Deep tear:

- Affects the medulla&cortex.
- This leads to hematuria.

4. Complete tear:

- Extends from the capsule to the renal pelvis.
- This leads to perinephric hematoma & hematuria.

5. Avulsion of a pole.**6. Avulsion of the pedicle.****7. Subcapsular hematoma.****8. Associated injuries.****Clinical Picture****Symptoms**

- 1- History of trauma.
- 2- Pain in the flanks. It may be obscured by injury to other organs.
- 3- Hematuria: (***degree is not proportionate to severity***).
 - a. May be absent in:
 - Superficial tear.
 - Avulsed pedicle or ureter.
 - Clot retention.
 - Traumatic anuria.
- About 30% of vascular injuries are not associated with hematuria.
- 4- Oliguria due to hypovolemia and hypotension.
- 5- Retention of urine due to clots in the bladder.

Signs**A-General:****1. Shock:**

- Irritability, pallor.
- Sweating, subnormal temperature.
- Low B.P.
- Rapid weak pulse. etc.

2. Associated injuries: (e.g. fracture lower ribs $\pm$ pneumothorax)**B-Local:****Abdominal examination:**

1. Inspection: - Ecchymosis & bruises in the loin. -Fracture ribs. - Rigidity
2. Palpation:
 - Tenderness in the loin.
 - Loin swelling due to extravasation of blood & urine.
 - If the peritoneum is torn $\rightarrow$ diffuse abdominal tenderness and distention.
 - Manifestations of intraperitoneal hemorrhage (guarding, rebound tenderness).
3. Percussion: shifting dullness.
4. Auscultation: $\downarrow$ Intestinal sounds.

DRE: fullness in rectovesical pouch (or Douglas pouch in females.)

Rupture kidney may be retroperitoneal with minimal shock with no peritoneal irritation.

Complications

Early

1. **Anuria** → traumatic due to:
 - Shock. - Reflex inhibition of both kidneys. - Injured solitary kidney.
2. **Bad condition** → Hemorrhage & shock.
3. **Clot retention** → due to obstruction of the bladder outflow by clots.

Late

- 1- **Pseudohydronephrosis** few weeks after injury due to:
 - Deep tear in the kidney + ureteric obstruction.
- 2- **Perinephric abscess.**
- 3- **Nephroptosis** due to tearing of the supporting tissues.
- 4- **Hypertension** after few months from renal fibrosis.
- 5- **Renal artery aneurysm, A-V fistula.**
- 6- **Hydronephrosis** due to fibrosis around the kidney leading to ureteric obstruction.
- 7- **Renal atrophy or fibrosis.**

Investigations

Better to resuscitate the patient 1st (anti-shock measures)
→ Warmth, oxygen, 2 central IV line, fluids or blood transfusion & Vit K.

For diagnosis

1. **U/S:** perinephric haematoma and/or free blood.
2. **CT scan:** if doubtful diagnosis
3. **Angiography or Tc scan:** for vascular or minimal injuries
4. **Plain x-ray:**
 - Shows any foreign body, fracture ribs or associated hemothorax.
 - Obliterated psoas shadow from the hematoma.
5. **I.V.U infusion urography**
 - Done to visualize the upper urinary tract as soon as the shock is controlled & the blood pressure is above 100mm Hg. It may show :
 - Normal function & configuration of kidney if the injury is minimal.
 - Deformed renal pelvis or calyces if there is laceration or blood clots.
 - Extravasation of contrast within the renal shadow or intoperirenal space.
 - Non visualization of kidney due to total pedicle avulsion , arterial thrombosis or severe contusion causing vascular spasm.
 - Confirms the presence of a functioning kidney on the opposite side , as nephrectomy of the injured kidney may be needed.

For complications

1. Urine analysis → RBCs, etc.....
2. KFTs.
3. CBC, FBS.

Treatment

- **General** → If the patient is poly-traumatized: ABCDE
→ Treatment of shock.
- **Specific** → Conservative management.
→ Surgical management if indicated.
- Treatment of associated injuries and other complications.

A. Conservative treatment

- ▣ Hospitalisation with bed rest until hematuria has ceased & local signs of injury have subsided.
- ▣ Analgesics for pain.
- ▣ Large fluid intake to guard against clot retention & for hypovolemia.
- ▣ Broad spectrum antibiotics to guard against secondary infection.
- ▣ Follow up parameters
 - Pulse, blood pressure, & size of any perirenal mass.
 - Repeated samples of urine are examined & compared grossly for red color.
 - Haemoglobin & haematocrit estimations.
 - Repeated urine analysis for RBCs.

B. Surgery**► Indication**

1. Persistent progressive hematuria or failure to stabilize vital signs.
2. Presence of a progressively enlarging perirenal mass.
3. Evidence of perirenal infection.
4. Penetrating injuries.
5. Renal pedicle injury (5% of all injuries).
6. Presence of an associated intraperitoneal injury.
7. Indications for delayed surgery
 - If hydronephrosis develops, it is treated by relief of obstruction.
 - If hypertension develops, vascular repair or nephrectomy is performed.

► Principles

1. Transperitoneal approach.
2. A hematoma around the injured kidney is evacuated.
3. Traumatized & devascularized renal tissue is debrided.
4. Small defects of cortical tissue are approximated by sutures. Large defects are filled by omental or perirenal fat to obliterate dead space.
5. Water-tight closure of the pelvicalyceal system.
6. Partial nephrectomy is done if one pole of the kidney is avulsed.
7. Nephrectomy is done if the kidney is shattered or with complete avulsion of vascular pedicle, provided that the other kidney is functioning well.

Injuries of the bladder

⇒ Types:

1. Intraperitoneal rupture of the bladder.
2. Extraperitoneal rupture of the bladder (DD → intrapelvic rupture of urethra)

	Intraperitoneal rupture (20%)	Extraperitoneal rupture (80%)
Causes	- Occurs in males - Full bladder & direct trauma applied to suprapubic region.	- More in males. - Bladder is injured 2ry to fracture pelvis
Pathology	- Urine escapes into peritoneal cavity → peritonitis in the area - Rupture usually occurs between the roof and post wall of the bladder	- Urine extravasates into the perivesical & periprostatic spaces, retropubic space (cave of Retzius) and then ascends up between peritoneum & fascia transversalis.
Symptoms	▪ History of trauma. ▪ Suprapubic pain. ▪ Urine retention with NO desire to micturate in intra-peritoneal type, but the desire is preserved in extra-peritoneal type. ▪ Hematuria.	
Signs	▪ General: shock ▪ Local: Inspection: bruises, ecchymosis & rigidity Palpation: guarding, rebound tenderness Percussion: shifting dullness Auscultation: diminished intestinal sounds DRE: fullness in rectovesical pouch Catheter can be passed easily & no urine is obtained (only blood)	▪ General: shock. ▪ Local: Inspection: bruises, ecchymosis & rigidity in suprapubic region due to associated fracture pelvis Palpation: guarding, rebound tenderness in suprapubic region Percussion: no shifting dullness DRE: empty rectovesical pouch Catheter can be passed easily & blood + small amount of urine
Complications	• Pelvic abscess from infection of hematoma or urine collected. • Delayed peritonitis. • Partial incontinence if bladder neck is injured.	
Investigations	1-X-ray	
	Ground glass appearance (due to urine in the lower part of the abdomen).	Fracture pelvis.
	2-U/S:	
	Free fluid in peritoneum	
	3- Ascending cystography or IVU	
	Leaking of the dye from the urinary bladder	

Treatment

- If the patient is polytraumatized:
 - ABCD + primary survey + treatment of life threatening injuries
- Treatment of shock: (I.V fluids & blood).
- Treatment of associated injuries: if present
- Treatment of ruptured bladder:
 - Through a **midline** suprapubic incision.
 - The extravasated urine is evacuated.
 - Expose the bladder & the bladder is sutured with a single layer of 2/0 polygalactin 910.
 - Placement of a suprapubic drain, retropubic drain and urethral catheter.

N.B. The fractured pelvis should not be treated by internal fixation in the presence of urine extravasation for fear of osteomyelitis.

Rupture of the male urethra

Types

According to site

1. **Extra-pelvic** (anterior, bulbous or penile urethra).
2. **Intra-pelvic** (posterior or membranous urethra) → D.D extraperitoneal rupture of bladder

According to extent

1. Complete.
2. Incomplete.

Urosurgery

	Extrapelvic rupture (most common)	Intrapelvic rupture
Cause	Trauma to perineum (Falling astride or kick).	Try to fracture pelvis
Pathology	• The tear may be partial or complete. • The urine collects under Camper's, Scarpa's & Colle's fasciae at the penis, perineum, scrotum, anterior abdominal wall & then descends down to the upper part of the thigh.	• Tear of the urethra, which is usually complete. • Avulsion of the pubo-prostatic ligament → floating prostate. • Extravasation of urine (in the cave of Retzius)
Symptoms	Severe pain in perineum. • History of trauma. • Urine retention with desire to micturate. • Urethral bleeding. • The triad of urethral hemorrhage, perineal hematoma and retention of urine is diagnostic of extra-pelvic rupture.	Severe pain in hypogastrium.
Signs	▪ General: Shock. ▪ Local: - Perineal hematoma - Bleeding per urethra. - Bladder is full. - Catheter is never used if urethral injury is suspected.	▪ General: Shock. ▪ Local: - Fracture pelvis - Bleeding per urethra. - Bladder is full. - Catheter is never used if urethral injury is suspected. PR: prostate can not be felt.

Investigations	Plain X-ray	
	-ve	Fracture pelvis
Treatment	▪ Urethrogram: - Extravasation of the dye ▪ Cystogram: - For associated UB injury.	
	1. If the patient is polytraumatized (mainly in intrapelvic type): ABCD + primary survey + treatment of life threatening injuries 2. Treatment of shock (IV fluids & blood). 3. Treatment of associated injuries if present 4. Suprapubic cystostomy. 5. Later on, urethral dilatation.	

Renal Tumors

		<u>Wilm's Tumor</u> (<u>Neohroblastoma</u>)	<u>Hypernephroma</u> (Renal adenocarcinoma)
Incidence		10% of childhood tumors (The 4 th common abdominal malignancy in children)	80% of renal tumors (The most common renal parenchymal tumor)
		Deletion of chromosome 11 in familial form	Deletion of short arm of chromosome 3
Etiology		usually below 4 years	usually between 50-70 years
		More in males	More in males
Pathology	Site	may be bilateral 6%	Usually unilateral (bilateral in 2%)
	Origin	Embryonic mesodermal cells (meso- & meta-nephric remnants)	Epithelium of renal tubules N.B. Hypernephroma is a <i>misnomer</i>
	Macro	- Solitary sharply demarcated encapsulated mass, areas of hemorrhage & necrosis + grayish or pinkish white + early invasion of the capsule, while invasion of the pelvis is late .	Mass (mainly from upper pole of kidney) infiltrating edge, area of hge& necrosis + <u>golden yellow color</u> + <u>usually invade the pelvis early</u> .
	Micro	<u>Mixed tumor</u> : It's differentiated in 80% of cases (of good prognosis) OR anaplastic in 20% of cases (of bad prognosis)	<u>Adenocarcinoma</u> a. Cells: - Forming acini or sheets + signs of mitosis & loss of polarity. - Clear cells (due to lipid, glycogen or cholesterol content) or granular cells. b. Vascular CT.

Staging

- I Confined to renal parenchyma.
- II Invade → Perinephric fat
- III perinephric fascia
- IV (a) Invade adjacent structures
(b) Distal metastasis
- V Bilateral

- a. Stage I → Tumor confined to the kidney.
- b. Stage II → Tumor confined to Gerota's fascia and involves the perinephric fat.
- c. Stage III → Tumor involves the renal vein and/or regional L.Ns.
- d. Stage IV → Distant metastases.

Spread

Direct spread to capsule early, pelvis late.

Direct spread to pelvis early, late, capsule

Lymphatic to para-aortic lymph nodes, Virchow's LN (by retrograde lymphatic spread).

Blood spread
Early to lungs.

Blood spread
2. Lung (canon ball in the X-ray).
3. Liver, bone.
4. Within the renal vein & IVC like **finger in gloves** → 2ry varicocele.

Clinical picture

- 1- The main presentation (90%) is an abdominal mass: which is smooth, firm & is confined to one side of the abdomen.
- 2- vague abdominal pain in 30% of cases (d.t. associated minor trauma & hemorrhage within the tumor).
- 3- **Microscopic** hematuria occurs in 50% of cases.
- 4- Hypertension is present in up to 60% of cases.
- 5- **Associated syndromes in familial type.**

- 1- **Hematuria:** present in 50% of cases which is painless, recurrent, profuse & total.
- 2- **Pain:** present in 40% of patients. It may be due to :
 - Stretch of the renal capsule by the neoplasm.
 - Passage of blood clots causing ureteric colic.
 - Infiltration of adjacent lumbar nerves causing referred pain.
- 3- **Renal mass:** Irregular, hard, renal swelling in 30% of cases (in advanced stages).
- 4- **Abnormal presentation as:**
 - a-Secondary varicocele:** rapidly enlarging varicocele which does not empty on elevation of the scrotum.
 - b- Metastasis:** May be the first presentation e.g. Pulmonary deposits.
 - c- FUO.**
 - d- Para-malignant syndrome** (e.g. hypercalcemia, polycythemia HTN & Cushing)

D.D

(of abdominal swelling in child):-

1. Neuroblastoma
2. Infantile poly-cystic kidney.
3. Congenital hydro-nephrosis.
4. Hepatoblastoma (on Rt. Side)
5. Splenomegaly (on Lt. Side)

Investigations**For diagnosis:**

1. CT scan (spiral)
2. U/S → abdominopelvic
3. Plain urinary tract (PUT): soft tissue shadow with cresenteric calcification
4. IVU → displaced pelvi-calyceal system (rarely invaded)
5. Ascending pyelogram to diagnose early changes in the pelvis.
6. Renal angiography to differentiate cysts from solid swelling (now replaced by CT)
7. Urine analysis → hematuria

For staging

1. CXR.
2. Bone scan.
3. Pelviabdominal U/S.

For preoperative preparation

1. CBC → anemia, high ESR.
2. KFTS, LFTS, FBS, CXR, ECG

Treatment**Surgery**

- For operable tumors → radical nephrectomy.
- For large non-operable tumours →
A course of preoperative chemotherapy usually shrinks the tumor, which can then be removed.

D.D:-**A. Of painless hematuria.**

- 1- TCC of urinary tract
- 2- T.B.
- 3- Glomerulonephritis.

B. Of renal swelling in adult:-

1. Solitary cyst
2. Adult poly-cystic kidney.
3. Hydro-nephrosis
4. Splenomegaly (on Lt. side)
5. Hepatoblastoma (on Rt. Side)

For diagnosis:

1. **CT scan (spiral)**
2. **U/S (abdomino-pelvic)**
3. **Plain urinary tract (PUT) →** soft tissue shadow with obliterated psoas shadow.
4. **IVU →** pelvi-calyceal system is Dilated, Enlarged Amputated & Distorted (**DEAD**) → (**Irregular** spider leg deformity)
5. **Ascending pyelogram** to diagnose early changes in the pelvis.
6. **Renal angiography** differentiates cysts from solid swellings (now replaced by CT)
7. Urine analysis → hematuria.

For staging

- CT scan, bone scan.
- U/S (abdominopelvic).
- CXR: cannon ball metastasis.

For preoperative preparation

- CBC → anemia, polycythaemia (4%), high ESR.
- KFTs, LFTs, FBS, ECG & CXR.

-For early operable cases (stages I & II) → Radical nephrectomy (removal of the kidney within its sheath of Gerota's fascia + the ipsilateral adrenal gland) via transperitoneal approach.

-For inoperable cases → Palliative treatment e.g. analgesics, irradiation & immunotherapy using alpha or gamma interferons & interleukin-2.

Prognosis	Post-operative treatment ■ If there is no residual tumor → adjuvant chemotherapy, using actinomycin D, vincristin & adriamycin. ■ If there is residual tumor → radiotherapy is added for local control.	
	■ Chemo & radiotherapy have improved the overall prognosis to 80% 5-year survival. Early cases are usually cured.	
NB. If invaded renal v. or IVC → open & excise the tumor. If solitary metastasis → excision e.g. pneumonectomy (for cannon ball)		

Carcinoma of UB

	<u>SCC</u>	<u>TCC</u>
Pathology		
Age	20-40	> 60
Sex	♂: ♀ → 4: 1	♂: ♀ → 3: 1
PF	1. Chronic irritation by bilharzial ova or stone. 2. Urinary metabolites. 3. Chronic infection. 4. precancerous lesions: (histopathological) ■ Brunns nests. ■ Cystitis cystica. ■ Vesical leukoplakia. ■ Squamous metaplasia.	1- Industrial carcinogens: ■ Aniline dye, rubber 2- Cigarette smoking 3- Pelvic irradiation 4- Cyclophosphamide. 5- Genetic factors as p53 gene mutation.
Site	Usually at posterior & lateral walls	Usually at the base and around ureteric orificies.
Macro	Fungating mass (common) Malignant ulcer Infiltrating mass (rare)	Papillary mass or papillae, Ulcer. Nodule.
Micro	Cell nests if well differentiated	TCC is classified into: 1. Superficial TCC (75%): 2. Muscle invasive type (25%) 3. Carcinoma in situ (CIS) (associated in 5% of the cases)

Clinical picture

Symptoms

- ▶ The patient usually presents with exacerbation of symptoms of cystitis.
- 1- **Hematuria: (terminal)**
 - Does not attract the attention of patient in cases of SCC on top of bilharziasis & the hematuria is **painless** in cases of TCC.
- 2- **Necroturia:** passage of pieces of whitish tissue (fish odour)
- 3- **Frequency and dysuria:**
 - Commonly occur with muscle invasion.
 - With CIS, symptoms may be very severe (malignant cystitis).
- 4- **Pain (late):**
 - Deep seated pelvic or suprapubic pain (usually with muscle invasion).
 - Sciatica (with pelvic wall invasion).
 - Loin pain due to ureteric obstruction.
- 5- **Abnormal presentations:**
 - Cachexia, **anemia**, metastasis, **repeated attacks of lower UTI**, retention of urine by a pedunculated tumor or a clot, late → uremia.

Signs

- a. **General:**
 - Manifestations of complications.
 - Cachexia, anemia, metastasis, fever if infected, late uremia.
- b. **Local**
 - Bimanual examination under general anesthesia (better done under anesthesia) to assess local spread.

Complications

Spread

- ⇒ **Direct spread:** to pelvic structures e.g. prostate in male, vagina or uterus in females, rectum, etc.....
- ⇒ **Blood spread:** very rare & late to the lungs, liver & bones
- ⇒ **Lymphatic spread:**
 - Perivesical LNs → internal iliac LNs → common iliac LNs → para-aortic LNs → thoracic duct → Virchow's.

Lymphatic spread in SCC on top of bilharziasis is delayed due to fibrosis & calcification in the bladder & peri-vesical tissue & local LNs

Others (in order of frequency)

- Ulceration. - Hemorrhage. - Infection. - Fistula formation.
- Urinary tract obstruction → hydronephrosis up to renal failure.

DD

- Urinary bladder stone. - Cystitis.

Staging

Wallace staging of bladder cancer is based on bimanual examination.

- T 0 → no palpable mass**
- T1 → Palpable mobile mass, but no induration of bladder wall**
- T2 → Palpable mass with induration of bladder wall.**
- T3 → Palpable mobile wall, with extravesical spread**
- T4 → fixed bladder mass**

Investigations

For diagnosis

1. **Cystourethroscopy + multiple biopsies:** (from the tumor base & adjacent mucosa)
2. **IVU:**
 - Shows irregular filling defect
 - May show manifestations of back pressure (hydroureter, hydronephrosis)
3. **Ascending cystography:** if IVU is contraindicated (e.g. uremia)
4. **CT & U/S.**
5. **Urine analysis:**
 - Recently: nuclear matrix protein (NMP22) for screening.
 - Hematuria, necroturia, pyuria. - Culture & sensitivity.
 - Cytology (shows Bilharzial ova, RBCs >5 /HPF, malignant cells).

For staging

1. CXR.
2. Bone scan.

For preoperative preparation

1. CBC → anemia, increased ESR.
2. K.F.Ts → uremia (in late cases).
3. LFTs → elevated ALP in metastasis.
4. FBS, CXR, ECG.

Treatment (according to type)

SCC (radio and chemoresistant)

Radical cystectomy + urinary diversion (best by ileal conduit)

TCC (according to the type and stage)

⇒ Superficial bladder cancer and CIS:

- a. Transurethral resection (TUR) + multiple biopsies (to detect unsuspected CIS and muscle invasion).
- b. Intra vesical chemotherapy (Mitomycin C) or immunotherapy (BCG).
- c. Follow up by regular cystoscopies every 3 months.

⇒ Muscle invasive type:

- a. Surgical:
 - 1-if no LN involvement → radical cystectomy+ urinary diversion (best by ileal conduit)
 - 2-if LN involvement → partial cystectomy (palliative).
- b. Radiotherapy.
- c. Chemotherapy (methotrexate, vinblastine, adriamycin & cisplatin) → MVAC.

Prostatic Enlargement

	BPH	Carcinoma of Prostate
Incidence	♂ > 50 years	commonest cancer in ♂ > 65 yr
Nature .	Benign condition (adenoma or hormonal imbalance).	Malignant.
Pathology		
Site .	Transitional zone (periurethral).	peripheral zone
Macro	- Fibrous trabeculae. - Yellowish	- Gritty sensation on cutting. - Greyish color
Micro	- Acini hyperplasia. - Corpora amylacia (dried prostatic secretions)	- Usually Adenocarcinoma. - Scirrhou carcinoma. - Rare anaplastic.
Complications		
Others	- Haematuria - Retention of urine - Renal failure - Back pressure - Bladder stone - Cystitis	- Haematuria - Retention of urine - Renal failure - Back pressure
Spread		<u>Direct</u> → pelvic organs <u>Lymphatic</u> External and Internal iliac LNs → common iliac Lns → para aortic LNs → thoracic duct → virchow's L.N. <u>Blood spread</u> Bone metastasis (usually osteosclerotic) Especially to lower vertebrae & pelvic bone.

Clinical Picture

Symptoms

- a) **Asymptomatic**
- b) **Prostatism**
 - 1- **Frequency**
(at night then by day & night)
 - 2- **Difficulty:**
To start, to maintain and to finish micturition.
 - 3- **Sexual symptoms:**
Increased libido at the start, later on impotence.
 - 4- **Symptoms of complications:** as retention.

- a- **Asymptomatic**
- b- **Prostatism**
(disturbance of function)
- c- **Pain** → (Perineal, Suprapubic)
- d- **Occult presentation** due to metastasis
- e- **Complications:** retention

Signs

- General** → Uremia, fever
Local
- mass or tenderness in renal angle (hydronephrosis)
 - Suprapubic palpable bladder.
 - **PR** → **soft, smooth, preserved** sulci & freely **mobile** rectal mucosa

- General** → Uremia, cachexia
Local
- mass or tenderness in renal angle.
 - Suprapubic palpable bladder.
DRE → **hard & nodular, lost** sulci & **fixed** rectal mucosa.

Investigations

A-For diagnosis:

1- Trans-rectal U/S:

Very accurate and non-invasive

The same

2- IVU

- Smooth filling defect in the bladder base.
- Hooking of the ureters on entering the bladder (fish-hook sign)
- Trabeculations, sacculations and diverticulae.
- Post voiding film → residual urine.
- Exclude gross pathology of the urinary tract.

- Irregular filling defect in the bladder base.
- May show back pressure effects as hydroureter & hydronephrosis.

3- PSA

< 15 ng/ml

- 15 ng/ml: is suggestive of □.
- 30 ng/ml: is diagnostic of □.

4- Cystourethroscopy + biopsy

- Adenoma
(It is indicated in patients presenting by hematuria to exclude bladder pathology)

- Adenocarcinoma

B- Others**For complications:***** Lab:**

- Urine analysis → C & S, hematuria.

- KFTs

*** Instrumental:**

- Uroflowmetry → if maximum flow rate is < 15 ml/sec → bladder neck obstruction.

For staging

- Bone scan (esp. if PSA > 20 ng/ml)

- X-ray spine → osteosclerotic or may be osteolytic lesion.

- CXR.

For preoperative preparation:

KFTs, LFTs, CBC, FBC, ECG

Treatment**⇒ For BPH:**

Asymptomatic cases → nothing

Mild non-complicated cases → conservative treatment in the form of:

1. Avoid factors which predispose to acute retention of urine.
2. Urinary antiseptic, alpha blocker & 5 alpha reductase inhibitor

Minimally invasive procedures:

- The aim of these procedures is temporary relief of BPH when definitive treatment is contraindicated in high risk patients.

▪ Methods:

- Thermotherapy. Microwave heat therapy.
- Endoscopic transurethral cryo-ablation of the prostate.
- Endoscopic transurethral prostatic stents.

▪ Indications for surgery

1. Severe prostatism
2. Complicated prostatism

▪ Methods:

1. T.U.R.P.
2. Transvesical prostatectomy (Freyer's)
3. Retro pubic prostatectomy (Millin's)
4. Perineal (young's)

NB:**Preoperative management:**

a- Antibiotics. b. Treatment of complications.

Postoperative management: irrigation of UB by a 3-ways catheter.

⇒ For cancer prostate:

Operable cases: → Total radical prostatectomy & external beam irradiation

Inoperable cases: → A combination of:

1. Hormonal treatment (see later).
2. Palliative transurethral resection.
3. Treatment of metastasis: e.g. bone metastasis → radiotherapy, internal fixation for pathological fractures.

Hormonal treatment:

Effect: → Inhibit androgen production.

Preparations:

→ LHRH agonist, antiandrogen: has less systemic side effects.

NB:

No treatment in cases of small well differentiated tumors in elderly men may be managed by watchful waiting, particularly if their life expectancy is less than 10 years.

Prognosis

- 1- With localized tumors to the prostate, the 10-year survival rate is 50%.
- 2- If metastasis is present, the 10-year survival rate drops to 10%

Hematuria

Definition

- Presence of blood in urine (always abnormal whatever its type)

Classification**1. According to the amount**

- Microscopic → blood is detected only chemically.
- Macroscopic → frank blood (red urine)

2. According to the presence of pain

- Painless (more common)
- Painful (the type of pain may help to identify the cause)

3. According to the relation to the urinary stream

- Initial hematuria: indicates urethral causes
- Terminal hematuria: indicates UB or prostatic causes
- Total hematuria: indicates renal or ureteric causes

Causes**a. General causes**

1. **Bleeding tendency:** e.g. pupura & hemophilia.
2. **Hypertension.**
3. **Drugs:** anticoagulants

b. Local causes

	Vascular	Infection	Trauma	Neoplasms	Others
Kidney	▪ Renal infraction	▪ Pyelonephritis ▪ TB	▪ Stone ▪ Rupture kidney	▪ Wilm's ▪ RCC ▪ TCC & SCC of the renal pelvis	▪ Polycystic kidney
Ureter		▪ TB	▪ Stone	▪ TCC of ureter	
UB		▪ Bilharziasis ▪ TB ▪ Non specific cystitis	▪ Stone ▪ Rupture bladder	▪ TCC ▪ SCC ▪ Adenocarcinoma	
Urethra		▪ Urethritis	▪ Stone ▪ Rupture urethra	▪ Urethral carcinoma	
Prostate		▪ Prostatitis		▪ Cancer prostate	▪ BPH

The commonest causes are: stones, bilharziasis, BPH, hypernephroma & trauma

1. Stones

- **Type of patient:** middle aged male
- **Symptoms:**
 1. Painful hematuria (terminal, initial or total according to the site)
 2. Pain:
 - Dull aching pain in the flanks if renal stone.
 - Ureteric colic if ureteric stone (severe agonizing loin to groin pain usually with nausea & vomiting)
 - Suprapubic pain with dysuria, strangury & frequency if bladder stone.
 - Urethral pain referred to the tip of penis if urethral stone.
- **Signs:**
 1. General: - Fever if infection occurred. - Uremia
 2. Local: hydronephrosis → tender loin mass
- **Complications:**
 1. Acute retention (in bladder or urethral stone) → painful desire to micturate & tender enlargement of the bladder.
 2. Calculus anuria (in complete ureteric obstruction if bilateral or unilateral in the only functioning kidney) → no urine, no desire and empty bladder.

2. RCC

- **Type of patient:** male, 40 years
- **Typical presentation:**
 1. Hematuria: painless, periodic, persistent, total.
 2. Late: loin pain or clot colic, mass in the flank.
- **Atypical presentations:**
 1. Cachexia, metastasis (cough, hemoptysis)
 2. Secondary varicocele
 3. Paraneoplastic \$ as: polycythemia, hypercalcemia, cushing disease.

3. Bilharzial cystitis

1. History of bilharziasis (endemic area)
 2. Painless terminal hematuria with anemic manifestations as: pallor & easy fatigability
 3. Pyuria and dysuria due to repeated infections
- **Complications:** as stones, hydronephrosis, bladder SCC & late → uremia.

4. BPH

- **Type of patient:** male > 60 years
- **Symptoms:**
 1. Prostatism (LUTs):
 - Frequency, 1st nocturnal then day & night.
 - Hesitancy, intermittent flow, post-micturition dribbling.
 - Sexual: increase libido then impotence
 2. Complications: as acute retention with alcohol or withholding urine
- **Signs:**
 1. Non-tender bladder enlargement ± tender lion mass.
 2. DRE → smooth, soft, symmetrical, preserved sulci & mobile rectal mucosa

Investigations

(Cystoscopy is the most important one, better during the attack)

1- Laboratory

1. **Urine analysis:** bilharzial ova, RBCs, pus cells, malignant cells
2. **KFTs:** blood urea & serum creatinine.
3. **Tumor markers:** as PSA for prostatic causes.

2- Radiological

- 1) **Plain X-ray:** stone, TB, Bilharziasis.
- 2) **U/S:** stones, tumor, congenital polycystic kidney.
- 3) **IVU:** → Stones, tumors (filling defect).
→ SEP (smooth elevation of bladder neck).
→ Hypernephroma (DEAD of pelvicalyceal system).
- 4) **Ascending cystography:** carcinoma of UB, diverticulum.

3- Instrumental

Cystoscopy:

- a. With biopsy from any lesion.
- b. If bleeding is unilateral → ureteric catheterization & do ascending pyelography

Treatment

(Not mentioned if the question is DD of hematuria)

1. **Anti-shock measures** (IV fluids, blood transfusion....)
2. **Stop bleeding** by: vitamin K injection, dicinone & cyclokapron IM
3. **Treatment of the cause-** e.g.
 1. **Stone:**
 - a. < 1 cm. → conservative.
 - b. 1-2 cm. & not impacted.
 - Pelvis & upper ureter → ESWL
 - Lower ureter & bladder → endoscopic extraction
 - c. > 2 cm. or impacted → endoscopic extraction
 - d. If failed above measures → open surgery according to the site.
 2. **SEP:**
 - a. TURP (if small).
 - b. Open prostatectomy (if large).
 3. **Hyper-nephroma** → radical nephrectomy.

Other causes of red urine

1. Certain food (beet root)
2. Certain drugs: carmurit, rifampicin.
3. Haemoglobinuria (hemolytic anemia), myoglobinuria.
4. Bilirubinuria (OJ).
5. During menstruation.

How to perform 3 glass test

Ask the patient to pass urine:

- The first part of urine in a glass.
 - The midpart in another glass.
 - The last part in a 3rd glass.
- a) Initial haematuria is urethral in origin.
 - b) Terminal haematuria is UB or prostate in origin.
 - c) Total haematuria is renal in origin.

Hypospadias

Definition

- A congenital anomaly of the urethra in which the urethra opens on the under surface of penis or perineum.

Incidence

- **The commonest congenital anomaly of urethra.**
- Positive family history in 8% of cases.

Etiology

(Embryological)

1. **Glanular type:** occurs due to failure of recanalisation at the glans of penis.
2. **Penile type:** occurs due to failure of fusion of inner urethral folds.
3. **Perineal type:** occurs due to failure of development of whole penile urethra.

Types

1. **Glanular type (commonest)** → EUM opens on under surface of the glans.
2. **Coronal. Meatus is at the coronal sulcus.**
3. **Penile type:**
 - EUM opens on the under surface of the shaft of the penis. It may be:
 - A- Anterior penile.
 - B- Mid-penile.
 - C- Posterior penile (peno-scrotal).
 - The distal part of urethra (corpus spongiosum distal to the urethral opening) is replaced by fibrous band.
 - This fibrous band is known as **urethral chordee**.
 - It causes curving of the penis downward on erection.
4. **Penoscrotal.**
5. **Perineal type:**
 - The scrotum is split.
 - The urethra opens between its 2 halves.
 - The penis is rudimentary.
 - The testis may be undescended.

Hypospadias is anterior in 65% of cases, midpenile in 15%, and penoscrotal or perineal in 20% of cases.

Clinical Picture

⇒ Depends on age of patient and severity of disease:

- **At birth:**
 - Abnormal prepuce present dorsally only (**hooded prepuce**).
 - Urethral opening more proximal than usual.
- **2-10 years:** wetting the clothes in micturition (abnormal stream).
- **10% of patients have inguinal hernia or undescended testis.**
- **8% of patients have upper urinary tract anomalies.**
- **After puberty:**
 - Bowing of penis downwards during erection due to presence of fibrous chordee.

Glanular type is a cosmetic & psychological problem, while other types will impair the function.

Complications

- Psychological distress.
- Pseudo infertility.
- Dysparunea.
- Associated hormonal manifestations (rare).

Investigations

- a. For associated conditions:
 - Hormonal assay and karyotyping (if bilateral undescended testes with hypospadias)
- b. To assess success of surgery:
 - Ascending urethrogram postoperatively.

Treatment

Circumcision should be avoided in all cases of hypospadias as the skin of the prepuce may be used in the repair later on.

- ⇒ Timing: at 6-18 months → improve the emotional and psychic results.
- ⇒ Aim:
 - Straight urine stream → psychological and social concern.
 - Correction of bowing of the penis → correct sexual function.
- ⇒ Principle:
 - 1- Removal of the chordee to correct the ventral curvature.
 - 2- 2-Urethral reconstruction using skin from the prepuce or penile skin.
 - 3- Circumcision after that.

Epispadias

Definition

- A congenital anomaly of the urethra in which the urethra open on the dorsal surface of penis.

Pathology

1. Incomplete type:
 - External urethral meatus (EUM) is situated on the dorsal surface of penis.
 - The patient is continent.
2. Complete type:
 - The condition is associated with ectopia vesicae.
 - The patient is incontinent.

Treatment

1. Incomplete type: as hypospadias but on the dorsum of the penis.
2. Complete type: as ectopia vesicae.

Ectopia Vesicae

Definition

A congenital anomaly characterized by absence of:

1. The lower part of the anterior abdominal wall.
2. The anterior bladder wall.

Incidence

- Male : female = 4 : 1

Etiology

1. Intrauterine rupture of UB through the anterior abdominal wall.
2. Failure of union between the 2 sides of the bladder & 2 sides of the abdominal wall.

Clinical picture

1. Absent anterior abdominal wall from the umbilicus downwards (umbilicus is absent)
2. Absent anterior bladder wall.
3. Widening of symphysis pubis → waddling gait.
4. Epispadias.
5. Rudimentary penis.
6. Bilateral undescended testes ($\pm$ bilateral inguinal herniae) with bifid scrotum.
7. The condition may be associated with distal anomalies as cleft lip, cleft palate, spina bifida etc...

Complications

1. Cystitis & Ascending pyelonephritis → death before 30 years.
2. Skin excoriation & ammoniacal odour.
3. Ulceration & bleeding from exposed bladder mucosa.
4. Chronic irritation of bladder mucosa → bladder carcinoma.

Investigations

- **Plain X-ray:** wide separation of the symphysis pubis.
- **IVU:** for associated urinary tract anomalies.

Treatment

Plastic reconstruction (in the 1st year of life)

1. Osteotomy of both iliac bones just lateral to sacroiliac joint.
2. Close the bladder $\pm$ augmentation (+ reconstruction of the bladder neck and sphincter or urinary diversion).
3. Closure of the abdominal wall which is helped with sacroiliostomy.
4. Later on; phalloplasty & urethral reconstruction.

Solitary Cyst of the Kidney

Etiology

- Not clear whether the lesion is congenital or acquired. Its origin may be similar to polycystic kidney or may be related to tubular obstruction.

Pathology

- **The cyst:**
 - Contains clear fluid & may contain altered blood.
 - Lined by flat epithelium.
 - Surrounded by a fibrous tissue & **compressed** renal tissue.

Clinical picture

- Usually asymptomatic.
- Dull aching **pain** in the loin due to stretch of renal capsule.
- A **swelling** may be felt in the loin.
- Complications (hemorrhage, rupture,.....)

Investigations

A- For Diagnosis:

1. U/S is very helpful:

2. IVU:

- Smooth amputation of a calyx.
- Localized smooth spider leg appearance.

3. Renal angiography: (not done now) → differentiate between cyst & tumor:

- Cyst → avascular.
- Tumor → abnormal vascularity.

The criteria of benign cyst?

Smooth wall, clear fluid, **no septa**, no malignant cell in aspirate, no residual mass after aspiration

B- For complications:

- Urine analysis: pus cells if infected.

Treatment

1. No treatment but follow up is required.
2. If the cyst cause hydronephrosis, treatment is aspiration of the cyst fluid and sclerosing by 95% alcohol. Marsupialization or excision may be required.
3. Atypical cyst (hemorrhagic, thick wall or cloudy fluid):
 - Percutaneous needle aspiration of content for analysis. Blood, high fat content or positive cytology gives high suspicion of malignancy.
 - Excise the extrarenal portion of the cyst (Kirwin's operation).
 - Partial nephrectomy may be considered.

Polycystic Kidney

Etiology

- Unknown cause → failure of fusion of mesonephric ducts with the metanephric ducts.
- It might be a part of cystic changes of the body (18% of patients have liver cysts, 30-40 % have intracranial aneurysms).

Pathology

- The kidney is enlarged with multiple cysts under the renal capsule.
- Cysts are not intercommunicated & are **not connected to the renal pelvis**.
- The cysts compress normal renal tissue → atrophy of tissue
- The cysts are lined by flattened epithelium.
- Condition is always bilateral but one side is more affected than the other.

Clinical Picture

Infantile type (A.R.)

- Still-birth with obstructed labor (huge-sized kidney).
- Infant may live for five months and then dies from uraemia or lung hypoplasia

Adult type(A.D.)

- + ve family history of the condition.
- First 10 years of life → no symptoms.
- 10 - 30 years → patient is asymptomatic but cysts could be demonstrated by U/S
- > 30 year:
 - Pain → dull-aching in flanks (stretch of capsule) or severe (due to complications as hemorrhage)
 - Swelling → loin.
 - Complications:
 - Hematuria (due to rupture of cyst in the pelvis).
 - Infection (due to stasis of urine).
 - Hypertension (75%).
- 40 - 50 year → renal function impairment.
- > 50 year → renal failure.

Investigations

1- For diagnosis

- U/S → **most accurate**.
- IVP → bilateral smooth spider leg appearance.

2- For complications

- KFT (for renal failure).
- Urine analysis → hematuria
- Plain x-ray → calcification

3- For screening (for other family members)

- U/S for family members above 20 years old.

Treatment

A-Treatment of congenital polycystic kidney:

1. **Medical:** to postpone uremia and delay renal transplantation
 - Decrease proteins in diet (prevention of uremia), ACE inhibitors (for hypertension), Antibiotics (for secondary bacterial infection)

2. **Surgical:** Rovsing operation:

- Puncture of cysts to diminish pain & pressure on the renal parenchyma.

- Nephrectomy is contraindicated except if renal transplantation is possible.

B- Treatment of complications:

Replacement therapy (for renal failure):

- Dialysis, kidney transplantation.

C- Genetic counseling:

- For newly married couples planning to have children.

Renal Tuberculosis

Etiology

- Organism: human TB (75%).
- Route: hematogenous.

Pathology

The kidney

- 1) The initial gross lesion is in the form of **small cortical TB foci**.
- 2) TB follicles affects renal papilla and pyramids then unite together to:
 - a. Burst into the pelvicalyceal system → **ulcero-cavernous type**.
 - b. Caseate in renal parenchyma → **caseo-cavernous type**.
- 3) Obstruction of the pelvis or ureter → **TB pyonephrosis**.
- 4) TB affection of perinephric tissues → **perinephric or cold abscess**.
- 5) Sometimes the kidney is completely destroyed and extensive calcification occurs (**auto-nephrectomy**).

The ureter

- Becomes thickened, fibrotic and shortened (Golf-hole appearance of ureteric orifice on cystoscopy)

The bladder

- Thickened, fibrosed, calcified and contracted with a capacity reaching 25 cc → Thimble bladder.

Genital lesions

- The seminal vesicles, prostate, epididymis, and the vas may become affected.

Clinical picture

Symptoms

- a. **General:** loss of appetite, loss of weight, night fever, night sweating, cough, expectoration & hemoptysis.
- b. **Local:**
 - **Pain:** sometimes dull aching in the loin or suprapubic in TB cystitis.
 - **Disturbance of function:**
 - **Frequency:** the earliest & main symptom; due to Irritation by TB debris, TB cystitis or contracted bladder.
 - **Pyuria:** opalescent urine.
 - **Hematuria:** transient; from ulceration of renal papillae.

Signs

- It is unusual for a tuberculous kidney to be palpable.
- **DRE:** for genital lesions.

Investigations

1. **CBC:** Leucopenia with relative lymphocytosis, Anemia, ESR (>100)
2. **Bacteriological:** early morning full urine sample sent for:
 - a- Urine analysis: sterile pyuria in acidic urine.
 - b- Ziehl Neelsen stain: Acid-fast alcohol fast bacilli.
 - c- Urine Culture on Lowenstein Jensen medium: or guinea pig inoculation
3. **Tuberculin tests.**
4. **CXR:** for pulmonary TB.
5. **Plain x-ray (KUB):** may show renal or bladder calcification (pseudo-calculi)
6. **IVU:**
 1. When calyx is affected it loses its clear outlines → Moth-eaten appearance.
 2. Small irregular bladder.
7. **Ascending pyelography:** minor changes in calyces.
8. **Cystoscopy.**
 - Shows ureteric and bladder affection (golf hole ureteric orifice, thimble bladder).

Treatment**Medical treatment**

- **Sanatorial** (sun, air, nutrition), good diet & vitamins.
- **Antituberculous drugs:** should be taken in combination.

Surgical treatment

- **Renal cavernotomy:** For single closed pyocalyx.
- **Nephro-uretrectomy:** in unilateral non-functioning kidney.
- **Ileo-cystoplasty:** augmentation of the contracted bladder by a loop of ileum.

Perinephric Abscess

Definition

- Pus collection around the kidney.

Etiology**Route of infection**

- **Blood born:** infection from a distal septic focus.
- **Direct spread:** of infection, usually from:
 - Appendix, GB, Pleura.
 - From nearby TB of vertebrae.
 - Pyonephrosis or renal cortical abscess.
- **Lymphatic spread:** periureteral lymphatic.

Causative organism

- Staph, strept.

Pathology

- Considered to be a subphrenic abscess.
- Unilocular or multilocular.

Clinical picture

Symptoms

1. **General:** hectic Fever, Anorexia, Headache & Malaise.
2. **Local:**
 - Pain: In the loin, increased by movement, breathing.
 - Swelling: in the loin.
 - Burning micturation if the kidney was the cause (rare)
 - Hiccough (due to irritation of the diaphragm)
3. **Symptoms of the cause:**
 - Burning micturation if due to renal infection.
 - Backache if on top of Pott's disease of the spine.

Signs

A-General: fever, tachycardia, pallor, sweating.

B-Local:

1. Inspection: - Rigidity in the loin. - Edema & reddening of overlying skin.
2. Palpation: tenderness & guarding.
3. Percussion: dullness.
4. Special sign: **flexion of the hip & painful extension (psoas spasm).**

Investigations

For diagnosis

1. **Laboratory:**
 - CBC: ↑ ESR, TLC is markedly raised (or leucopenia with relative lymphocytosis if TB)
 - ESR.
2. **Radiological:**
 - CT and U/S: are diagnostic & therapeutic (needle guided)
 - Plain X-ray: - Shows scoliosis of spine. - Obliteration of psoas shadow.
- Elevated fixed diaphragm.
 - IVU: shows loss of mobility of kidney in erect position or on lying down (Mathe's sign).

For the cause

- Plain X-ray spine for Pott's disease.

Treatment

Early

- Rest, 3As (analgesics, antipyretics and massive antibiotics. - Hot fomentations.

Late

- Drainage through catheter guided by U/S.
- If **multilocular or thick** → pus is drained through a lumbar incision.

Pyonephrosis

Definition

- Retention of infected urine and pus in the kidney.

Etiology

(Obstruction & infection)

- Organism: E.coli,...
- Route of infection: usually ascending infection.
- Predisposing factors: obstruction and ↓ immunity.

⇒ It may be:

1-Primary pyonephrosis → Infection then obstruction or occurs simultaneously.

2-Secondary pyonephrosis → Infection of pre-existing hydronephrosis
(Obstruction occurs at first → hydronephrosis, this is followed by infection)

Pathology

(usually unilateral: the renal parenchyma is destroyed & replaced by multiple cavities filled with pus.

A. Primary pyonephrosis

- Kidney not very much enlarged.
- Adhesions around the kidney.

B. Secondary pyonephrosis

- The kidney is hugely enlarged.

Clinical picture

A- Closed type

(In which no pyuria due to the obstructing agent & toxemia is severe)

- **General** → hectic fever, rigors, anorexia, headache, malaise, etc..
- **Local**
 1. Loin pain (throbbing) and tenderness.
 2. Renal swelling: usually small (large in 2ry pyonephrosis).

B- Open type

(The pus comes out in large amount so toxemia is less severe)

- Presents with triad of anemia, fever & renal swelling.
- With or without secondary cystitis (pyuria, frequency, burning micturation).

Complications

General

- Septicemia and septic shock in closed type.
- Renal failure.

Local

- Permanent renal scarring.
- Peri-nephric abscess.

DD

- Basal pneumonia & pleurisy.
- Acute cholecystitis.
- Acute appendicitis.
- Acute pancreatitis.
- Perinephric abscess.

Investigations

For diagnosis

- **CBC, ESR:** elevated TLC, high ESR.
- **Urine analysis, C&S:** pyuria in open type only.
- **U/S:** dilatation of the renal pelvis and calyces $\pm$ renal damage.
- **IVU:** (if KFTs within normal) after resolution of the acute attack $\rightarrow$ poor renal function and hydronephrosis on the affected side
- **Cystoscopy:**
 - 1- In open type: shows signs of chronic cystitis and purulent efflux from one ureteric orifice
 - 2- In closed type: ureteric catheter may be arrested at the site of obstruction, or may enter the pelvis and gives exit to purulent urine with marked relief of pain.

For cause

- **Plain X-ray:** may show stone.

For complications KFTs

Treatment

Treatment of pyonephrosis

Rest, 3 As (analgesics, antipyretics and parenteral antibiotics)

$\Rightarrow$ According to the type:

a- Open type:

Early (kidney is functioning): remove the cause of obstruction, antibiotics.

Late (kidney is not functioning):

- **Nephrectomy** $\rightarrow$ If the other kidney is with normal function.
- **Nephrostomy** $\rightarrow$ If the other kidney is with bad function, then After toxemia disappears:
 1. Treat the better function kidney.
 2. If kidneys are not yet functioning $\rightarrow$ dialysis and/or transplantation is done.

b- Closed type:

It is a surgical emergency:

- Drainage of the kidney by a ureteric catheter, nephrostomy tube or even open nephrostomy.
- Then after acute attack $\rightarrow$ treat it as open type.

Treatment of complications

- **Renal hypertension** $\rightarrow$ ACE inhibitors, nephrectomy of the affected kidney provided the other kidney is normal.
- **Renal failure** $\rightarrow$ replacement therapy (dialysis or transplantation).

Treatment of the cause e.g: proper management of stones .

Bilharziasis of UB

Etiology

- Due to *Schistosoma hematobium* (96%) or *mansoni* (4%).

Incidence

- Middle aged male (Male:Female = 3 : 1).

Pathology

Site

- The lesions affect the mucosa, muscle layer or peri-vesical tissue.

Macroscopically (cystoscopy) (Depending on duration, one or more can be seen)

1. **Bilharzial pseudo-tubercle:** (earliest specific appearance of the disease)
2. **Bilharzial nodule:** caused by fusion of pseudo-tubercles.
3. **Granuloma formation:** aggregation of nodules → masses.
4. **Bilharzial papilloma:** pedunculated masses.
5. **Ulceration (bilharzial ulcers):**
6. **Sandy patches:** calcified dead ova + degenerated overlying epithelium (more around ureteric orifices).
7. **Fibrosis:** resulting from 2nd infection → contracted bladder.

Microscopically

- Cystitis glandularis, cystitis cystica, leucoplakia → predispose to UB carcinoma.

Clinical picture

- Swimmer's or bather's itch due to penetration of cercaria through the skin may be followed 4- 12 weeks later by fever, urticaria & asthma.
- Such symptoms usually pass unnoticed until ova invade the bladder within 6 months after cercarial penetration.

Symptoms

1. Hematuria is the cardinal symptom. It is usually slight (few drops), & terminal.
2. Pain → Some urethral burning may be felt, but with the occurrence of ulceration, stone formation & malignancy. The pain is markedly increased.
3. Frequency.
4. Difficulty in micturition.

Signs

- No signs are elicited until complications supervene.

Investigations

For diagnosis

- **Urine analysis:**
 - Last few milliliters of early morning urine are taken for several consecutive days.
 - Bilharzial ova, RBCs, pus cells. Urine is usually alkaline (infected).
 - Negative results don't exclude bilharziasis.
- **Cystography:** to study the shape and the size of the bladder.
- **Cystoscopy:** to study the macroscopic pathology in the bladder wall + biopsy.
- **Antibody detection by ELISA.**

For complications

- **Plain x-ray & IVU:** shows bladder calcification and calcified lower ureter.

Treatment**Medical treatment**

- Praziquantel single dose 60mg/kg (max. 400mg) + urinary antiseptics (Antimony).

Surgical treatment

- Polyps: cystoscopic removal + biopsy.
- BNO: wedge excision to relieve urine outflow obstruction.
- Ulcer: cystoscopic cauterization

Imperfectly Descended Testis

A-Incompletely Descended Testis

Definition

- The testis is arrested at any site on the **normal** pathway to the scrotum.

Etiology

- ⇒ **Unilateral cases:** (*mechanical causes*)
 - Dysgenesis (small testis).
 - Short testicular artery.
 - Short spermatic cord.
 - Large testis.
 - Band of adhesions.
 - Associated hernial sac.
 - Inadequate inguinal canal or rings.
- ⇒ **Bilateral causes** (*hormonal causes or bilateral mechanical*)
 - It may lead to **Cryptorchidism** → hypogonadism
 - Deficiency of **maternal** HCG
 - Deficiency of **fetal** pituitary gonadotrophin (testis not sensitive to gonadotrophins).

Incidence

- 1% of all males and higher incidence in premature infants
- **Side:**
 - 50% right side (due to later descend of the right testis).
 - 30% left side.
 - 20% bilateral.
- Urinary tract anomalies in 13.5%.

The sites of arrest:

1. Intra-abdominal.
2. Inguinal canal (most common site).
3. Superficial inguinal pouch (very rare).

Complications

1. **Psychological.**
2. Liability to **trauma & torsion.**
3. **Epididymo-orchitis**
4. **Sterility:** *In bilateral cases (cryptorchidism)*
5. Liability to malignancy (**Seminoma**) 30 times more than normal.
6. **Indirect inguinal hernia.**

Clinical Picture

Symptoms

- **Mother** complains that one or both sides of her baby scrotum are empty.

Signs

1. **The Scrotum:**
 - Empty **poorly** developed scrotum (unilateral or bilateral).
 - The median scrotal raphe is deviated towards the affected side.
2. **The Testis:**
 - Usually present in the inguinal canal.
 - It is **not well developed.**
3. **Associated other anomalies (hypospadias)**

DD

1. Ectopic testis.
2. Retractable testis.
3. Surgically removed testis.
4. Agensis

Investigations

The most important investigation is laparoscopy

Radiological

1. **U/S & CT scan**
2. **IVP** for associated urinary anomalies

Instrumental

1. **Laparoscopy (the best).**

Laboratory

1. Karyotyping in the bilateral cases.
2. Hormonal assay.

Treatment**I. Treatment of incompletely descended testis:****A. Bilateral cases:**

1. **Hormonal therapy:** B-HCG 500U I.M. twice/wk for 6 wks.

N.B.:

- Never give B-HCG > 6 wks as testosterone secretion will occur.
 - Never give testosterone as it leads to short stature d.t. closure of epiphysis.
2. **Orchiopexy:** if failed hormonal therapy or adult cases but wait 6 months between The two sides.

B. Unilateral cases: Orchiopexy (bet. 6-24 months) by:

1. Place the testicles in Dartos pouch created between skin and Dartos muscle
2. If short Cord → Dissect the cord to elongate it by:
 - >> Fowler-Stein technique.
 - >> Two stage operation with 6 months interval.

II. Repair of associated hernia (if present)**B- Ectopic testis (Maldescended testis)****Definition**

- The testis has passed the external ring to an ectopic subcutaneous position.

Etiology

⇒ **Lockwood theory:**

- Rupture of the gubernaculum so one of the accessory tails is in work.

Sites**A. Around inguinal ligament:**

- **Above:** superficial inguinal pouch (commonest).
- **Below:** femoral triangle.

B. Around penis:

- **Above:** root of the penis.
- **Below:** in perineum.

C. Transverse scrotal.**Complications**

1. **Psychological.**
2. Liability to **trauma & torsion**

Clinical Picture**Symptoms**

- **Mother** complains that one or both sides of her baby scrotum are empty

Signs**1- The Scrotum:**

- Empty scrotum (unilateral or bilateral)
- Is well developed (normally, at the external ring the testis secretes hormones to develop the scrotal compartment of its same side.

2- The Testis:

- It is **outside** the inguinal canal & during muscle contraction the testis becomes better felt.
- It is of **normal size**.

DD

1. Arrested testis
2. Retractable testis.
3. Surgically removed testis.
4. Agensis.

Investigations

- No need for investigations as its site can be detected clinically.

Treatment

- Orchiopexy is easier as the cord is usually long.

C- Retractable Testis

Very mobile testis due to exaggerated cremasteric reflex it retracts with:

- Cold exposure.
- Scratch of the medial side of the thigh.

It is diagnosed by

1. Scrotum is well developed.
2. The testis is of normal size.
3. Repeated examination in warm room.
4. Make the child squat this help the descend of retractile testis, which is milked down to touch the scrotum floor.

Treatment: no treatment is required just reassurance.

Torsion of the Testis

No torsion on top of normal testis

Definition

- It is torsion of the testis and epididymis around the axis of spermatic cord presenting by a surgical emergency.

Etiology

Predisposing Factors

1. Imperfectly descended testis (incomplete or ectopic)
2. Inversion of the testis (commonest)
3. Long mesorchium.
4. High investment of the tunica vaginalis (Clapper in a bell effect).
5. Spirally attached cremasteric muscles.

Precipitating Factors

- Straining or minor trauma

Pathology

- Gangrene occurs within 8-12 hours if no treatment.

Clinical Picture

Type of patient

- Infant or adult 15-25 years with sudden severe testicular pain.

Symptoms

- Pain** → sudden severe agonizing pain in scrotum & lower abdomen.
- Swelling** → Testicular swelling.
- Reflex symptoms** → Nausea, vomiting & collapse.

Signs

⇒ **General:** pallor, sweating & tachycardia (up to shock).

⇒ **Local:**

1- Torsion of imperfectly descended testis:

- **The scrotum** is empty in the affected side.
- **The inguinal canal** is swollen, tender.

2- Torsion of the complete descended testis:

- **The scrotum:**
 - Swollen, red & tender.
 - Dimple in site of gubernaculum.
 - Elevation of the scrotum ↑ the pain.
- **The cord:** twist might be felt on the cord.
- **The testis is:**
 - High up in position.
 - Tender.
 - Small vaginal hydrocele.

DD (Of acute scrotum)**I. Acute epididymo-orchitis:**

	Testicular torsion	Acute epididymo-orchitis
Age	Adolescent & children	Adult or elderly
History	Mild trauma	UTI symptoms
Temperature	Slightly elevated	elevated
Scrotal elevation	↑ pain	Partially relieves pain
Urine analysis	Free	Pus cells
Doppler	Obstructed testicular Vs.	Patent Vs.

II. Strangulated inguinal hernia: irreducible, no impulse on cough, tense & tender**Investigations** Usually clinically diagnosed

- **Doppler & Duplex scan:** detect obstruction of blood flow in testicular vessels.
- **Urine analysis:** to exclude epididymo-orchitis.

Treatment

1. **Correct general condition.**
2. **Urgent operation:**
 - a- In early cases(viable testis):
 - Untwist the cord. - Orchiopexy.
 - b- In late cases (where the testis is gangrenous): orchiectomy
3. **Orchiopexy of the other testis:**

Testicular Neoplasm**Definition**

Abnormal rapid and invasive growth of malignant cells in the testis.

Incidence

- 99 % of testicular neoplasms are malignant.

Classification**A. Germ cell tumors:**

- Seminomas 40 %.
- Teratoma 32 %.
- Seminoma + Teratoma 14 %.

} Main types.

B. Interstitial tumors 1.5 %

- Leyding cell tumor.
- Sertoli cell tumor.

} Rare types.

C. Secondary tumors

- Lymphoma.
- Leukemic infiltration of the testis.
- Metastatic tumors.

Seminoma		Teratoma	
Age			
30 - 50 years.		20 - 35 years.	
Origin			
Seminiferous tubules.		Totipotent cells which have trigeminal potentiality.	
Risk factors of germ cell tumor			
- Undescended testis especially intraabdominal varities			
Pathology			
<u>Macroscopic picture</u> - Large & smooth surface - Homogenous & pink - Outline is not preserved (local infiltration) → so tuica can't be separated <u>Microscopically</u> sheets of Rounded or oval cells resembles spermatocytes, with lymphocytic infiltration		<u>Macroscopic picture</u> - Variable size&irregular surface - Heterogenous & yellowish - Outline is preserved → so tunica can be separated <u>Types</u> - Malignant teratoma anaplastica - Malignant teratoma intermedia (commonest type) - Teratoma differentiated (Dermoid) - Malignant teratomaTrophoblastica (choriocarcinoma)..	
Staging			
• Stage I → tumor is confined to the testis. • Stage II→ involvement of LNs below the diaphragm. • Stage III→ involvement of LNs above the diaphragm. • Stage IV→ distant metastasis.			
Spread			
• Local:Spermatic cord, epididymis, scrotal wall. • Lymphatic: paraortic lymph nodes → thoracic duct → Virchow's gland. • Blood (rare): early to the lungs are then to liver,bone&brain. <i>NB: If there is invasion of the scrotum the inguinal LNs will be affected.</i>			
Mainly lymphatic spread		Mainly blood spread	
Markers			
- B-HCG & LDH (Radio-sensitive)		- B-HCG & α-feto-protein (Chemo-sensitive)	

Clinical Picture

Type of patient

Middle aged male patient with testicular mass either discovered accidentally or after trauma as trauma arouses patient attention to mass

Symptoms

- May be **asymptomatic**.
- Painless testicular **swelling** (Gradual onset, progressive course) but late painful.
- Symptoms of metastasis for example bone pain.

Signs**1- Local:**• **Testis is:**

- Enlarged
- Hard, Heavy mass → later on areas of softening may be present
- May be fixed to surrounding structures in advanced cases.

• Early loss of testicular sensation.

• Secondary hydrocele (rapidly accumulating).

2- Abdominal: - Para aortic LNs enlargement. - Liver enlargement.**3- General:** manifestation of metastasis.**DD**

1. Syphilis (gumma).

2. Old clotted hematocle.

Investigations**For diagnosis****1. Radiological:**

- **Scrotal U/S:** shows testicular mass and calcification (is more frequent with seminoma).

2. Laboratory:⇒ **Tumor markers:**

- **Beta HCG (pregnancy test):** it is raised in teratoma & occasionally in seminoma
- **Alpha feto proteins:** raised in teratoma but never in seminoma alone.

3. Instrumental:**Frozen section biopsy****Indications:**

1. When clinical diagnosis is doubtful.
2. When the markers are -ve.

For staging

- **Bone scan, CT scan, liver U/S, CXR.**
- **IVP:** distortion of the ureteric course which may indicate para- aortic LN metastasis.

For preoperative preparation

- **CBC, blood sugar, ECG, urine analysis....**

Treatment**A- Initial treatment** is by high inguinal simple orchiectomy**B- Further management** depends on type & stage of the disease**Seminoma (radio- & chemo-sensitive)**

- **Stage I** → Radio-therapy to **para-aortic L.N.**
- **Stage II** → Radio-therapy is extended to **mediastinum**
- **Stage III** → chemo-therapy (cisplatin) +/- radiotherapy
- **Stage IV** → Chemo-therapy

Teratoma (radio-resistant)

- **Stage I** → Repeated assessment of the tumour markers.
- **Stage II-IV** → Combination chemo-therapy
(Cisplatin, methotrexate, bleomycin & vincristine)

Follow up

- Following surgery, follow up of patient with tumor markers must be done

Prognosis

- According to the type and stage of the tumor.

Varicocele

Definition

- It is varicosity (dilatation, elongation and tortuosity) of pampiniform & cremasteric plexuses of veins

Primary Varicocele

Incidence

- 15% of general population. Common in the Lt side.

Etiology**Predisposing factors**

- Age: Between puberty and 35 years
- Congenital weak mesenchyme:

Precipitating factors

- **Increase venous pressure**
 1. Prolonged standing.
 2. Straining as in constipation.
 3. Venous congestion due to unrelieved sexual excitement

Clinical Picture**Symptoms**

- Usually symptomless and discovered at medical examination but it may present by:
 - Pain:
 - Dragging pain: due to relaxation of Dartos & cremasteric ms.
 - Throbbing pain in case of thrombophlebitis.
 - Swelling: scrotal swelling.

Signs

- General:
 - Patient is usually tall & thin & visceroptosis may be present.
- Local:
 - Inspection:
 - Left side of the scrotum hangs lower than the right side.
 - Scrotal skin may show dilated veins.
 - Palpation
 - Scrotal neck test: fullness at the neck of the scrotum.
 - Varicosity is felt as a bag of worms.
 - Thrill on coughing due to turbulence of blood flow.
 - Swelling disappears when patient lies down & scrotum is elevated.
 - Small lax 2ry hydrocele → can be detected by pinching test.

To differentiate
it from 2ry
varicocele

Complications

1. Subfertility (20 % of cases)
2. Thrombosis → thrombophlebitis
3. Secondary hydrocele
4. Testicular atrophy (very late)
5. Neurosis

DD

- Inguino-scrotal swelling.

Investigations

Laboratory

- Semen analysis (for medico-legal importance) → **stress pattern**.

Radiological

- **Duplex scan** → detects reversed blood flow & bilaterality.

Treatment

Conservative treatment

(2 supports)

Scrotal support.

2- **psychological support**.

3- **Sedatives**.

4- **Avoid constipation**, pelvic congestion.

5- **Cold baths** to the scrotum.

Operative treatment

(**Varicoselectomy**)

○

Indications:

- Failure of medical treatment (Severe symptoms that can't be tolerated).

• **Complications:**

- 1- Subfertility.
- 2- Recurrent thrombophlebitis
- 3- Failure of medical commission.
- 4- Neurotic patient.

Secondary Varicocele

Causes

It is due to venous obstruction.

1. The commonest cause is **hypernephroma** (it spreads in the renal vein like fingers in glove).
2. Retroperitoneal tumors.
- 3- Retroperitoneal fibrosis.

It differs from primary type in

- 1- Older age group. (Usually >40 years)
- 2- Rapidly progressive.
- 3- No thrill on cough.
- 4- Size is not reduced when patient lies down & scrotum is elevated.

Hydrocele

Definition

- It is collection of serous fluid inside a part or the processus vaginalis.

Types

A. hydrocele of tunica vaginalis:

- 1- Congenital.
- 2- Infantile.
- 3- Vaginal hydrocele (1ry or 2ry)

B. Hydrocele of spermatic cord:

- 1- Encysted hydrocele of the cord
- 2- Diffuse hydrocele of the cord.
- 3- Hydrocele of hernial sac.

C. Rare types:

- 1- Hydrocele of canal of Nuck (in females).

Etiology

- 1- Excessive fluid formation as in secondary hydrocele.
- 2- Spermatic cord lymphatic obstruction by filariasis or low grade infection.

Complications

- 1- **Hernia of the hydrocele sac.**
- 2- **Hematocele.**
- 3- **Infection.**
- 4- **Rupture** usually traumatic but might be spontaneous
- 5- **Calcification.**
- 6- Bilateral huge cases might lead to **atrophy of the testis**. In unilateral cases
→no atrophy as hydrocele distends in wide scrotum.

Infantile Hydrocele	Congenital Hydrocele
Causes	
The sac has no connection with the peritoneum.	- It is due to failure of the obliteration of the processus vaginalis. - It connects with the peritoneal cavity. Small opening allow passage of fluid but not intestine.
Clinical Picture	
Symptoms - Swelling..... - There is no fluctuation in size	Symptoms - Mother reports that the baby has a swelling in the scrotum. - There is fluctuation in size by day and night.
Sings - Cystic, translucent inguinoscrotal swelling. - Not reducible manually.	
Treatment	
Eversion of the tunica	Excision of the upper part of the sac till the internal ring.

Primary Vaginal Hydrocele

Definition

- Exudate & specific gravity

Incidence

- Occurs in middle-aged males.

Causes

Its cause is unknown

Pathology

- Thin Amber yellow water, contains albumin , fibrinogen in concentration that resembles transudate , cholesterol in old standing cases

Clinical Picture

Symptoms

- Painless swelling in one of the scrotal compartment (gradual onset, progressive course, long duration)

Signs

- Inspection → Swelling in one scrotal compartment
- Palpation → non tender, cystic, translucent and purely scrotal swelling.

Investigations

- It is a clinical diagnosis.

Complications

As above

Treatment

1. Lord's operation (plication of tunica vaginalis)
2. Subtotal excision of the tunica vaginalis:

It is done in cases of:

- 1- Calcified tunica.
- 2- Loculated hydrocele.
- 3- Recurrent hydrocele after eversion of tunica

Other rare types of Hydrocele

Secondary Vaginal Hydrocele

Causes

- Disease in testis, cord, epididymis or post operative after repair of hernia.
- It is usually lax & rarely attains large size except in malignancy.
- If there is possibility to be with testicular tumor U/S, pregnancy test, alpha feto protein or even exploration and frozen section is done.

Encysted Hydrocele of the Cord

Causes

- Failure of obliteration of the middle portion of the processus vaginalis.
- It becomes distended with fluids.

Clinical Picture**Symptoms**

- Child or adult with painless swelling in the scrotal compartment.

Signs

- Cystic, painless, translucent swelling.
- Separated from the testis by interval.
- **Its mobility is restricted on downward traction of the testis. (traction test)**
- It is scrotal swelling.
- **It is mobile across the cord.**

Treatment

- Excision

Hematocele

Etiology

- Trauma
- Generalized bleeding disorders
- Tapping of hydrocele (commonest).

Complaint

- Swelling which is Tender, Bluish.

Fate

- 1- Absorbing is slowly & uncertain.
- 2- Clotted hematocele.
- 3- Infection.
- 4- Calcification.

Treatment

Exploration, evacuation, eversion of the tunica + cauterize the bleeder, & check out the testis for trauma or laceration.

Diseases of the Epididymis

Cysts of the Epididymis

Spermatocele**➤ Definition:**

- It is a retention cyst of the vasa efferentia.
- It containing living, dead sperms.
- **It contains Barly water fluids**
- **Fluid rich in cholesterol.**

➤ Symptoms:

- The patients usually complain of swelling in the scrotum (that he has a third testis).

➤ Signs:

- Painless cystic swelling at the upper pole of the testis.
- **Not separated from the testis by interval.** (to differentiate it from encysted hydrocele of the cord)

➤ Treatment:

- Excision for fear of complications.

Simple Cysts of the Epididymis

- It is related to vestigial structure (hydatid of Morgagni).
 - It contains crystal clear fluid, no sperms.
 - It may give multiple transillumination (Chinese lantern appearance).
- **Treatment** Excision only if causing complications.

Acute Epididymo-orchitis

Causative Organisms

- E.coli, Staph, Strepto, Gonococci.

Route of Infection

- 1- Along the vas.
- 2- Peri vasal Lymphatics.
- 3- Via blood stream.

Predisposing Factors UTI

Clinical Picture

Symptoms

- **Of the cause:** for example → dysuria
- **General:** FAHM.
- **Local:** Acute **painful** scrotal **swelling**, relieved by elevating scrotum.

Signs

- **General:** temperature 39° C
- **Local:**
 - ⇒ Scrotal skin is **red & edematous**.
 - ⇒ Enlarged **tender** epididymis.
 - ⇒ Secondary hydrocele.
 - ⇒ Signs of **pus loculus** if abscess is formed.

DD (Testicular torsion)

Complications

- 1- Testicular abscess
- 2- Testicular atrophy
- 3- Chronicity

Investigations

- 1- **Urine analysis** + culture & sensitivity
- 2- **Duplex** (to exclude testicular torsion).

Treatment

- 1- **Prophylactic:** proper treatment of UTI
- 2- **Curative:**
 - Antibiotics, analgesics & antipyretics.
 - Rest of the affected organ (elevation of the scrotum).
 - If an abscess formed = drainage.

Chronic Epididymorchitis

TB Epididymitis

Route of Spread

- It is usually secondary to urinary TB.

Pathology

- **If lymphatic borne:**
 - The tail is affected at first.
 - The **vas is thick & beaded** with tuberculous nodules.
- **In blood born:**
 - The head affected at first.
 - The vas remains unaffected.

Clinical Picture

Symptoms

1. Symptoms of TB toxemia.
2. Symptoms of urinary TB (see nephrology).

Signs

1. Signs of TB toxemia.
2. Epididymis is enlarged, firm with multiple sinuses discharging caseous material posteriorly.
3. DRE → TB nodules in the prostate and seminal vesicles.
4. Signs of urinary TB.

Investigations

- 1- Urine analysis → sterile pyuria.
- 2- IVU → to detect urinary TB.
- 3- Culture of secondary on Lowenstein media

Treatment

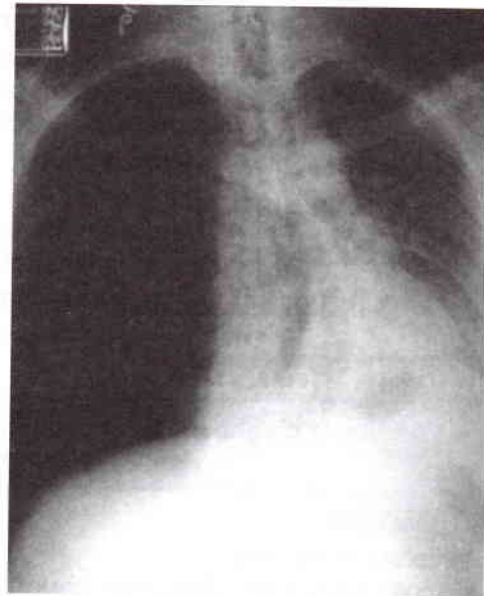
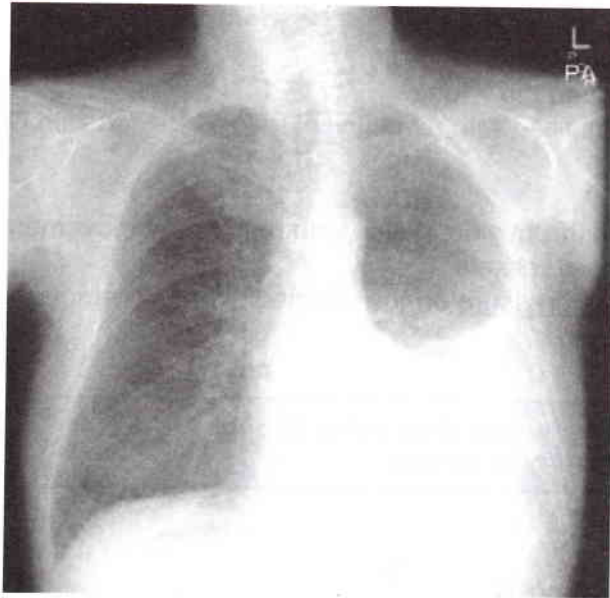
- 1- Improve the general condition (**Sanatorial treatment**).
- 2- **Anti - tuberculous drugs.**
 - If no response after 2 months:
 - We do epididymovasectomy.
 - Anti tuberculous drugs are given for another 4-6 months.

DD of inguinoscrotal swelling

► See D.D BOOK

7

CARDIOTHORACIC SURGERY



Pneumothorax

► **Definition:** Air inside the pleural cavity.

► **Etiology :**

- **Traumatic:**

- **Open injury:** e.g. stab wound.
- **Closed injury:** e.g. fracture rib.
- **Iatrogenic:** e.g. +ve pressure ventilation (Barotrauma).

- **Spontaneous:** e.g. rupture of emphysematous bullae, lung cyst or T.B. cavity.

Open Pneumothorax

Definition

- Air inside the pleural cavity d.t. sucking chest wound which allow free entry & exit of air from pleural space.

Pathology

1. **Respiratory failure (impairment of gas exchange) due to:**

i) Paradoxical respiration: on the affected side which:

- **Expands** slightly during **expiration**.
- **Collapses** more during **inspiration**.

ii) Pendulum respiration due to:

- Oscillation of air between the 2 lungs
(Normal lung is always filled with air deficient in O₂ & loaded with CO₂).

2. **Circulatory failure due to:**

i) Mediastinal flutter:

- The mediastinum moves from side to side with respiratory movements leading to kinking of great vessels

ii) Loss of negative intrathoracic pressure on the affected side → ↓ venous return → ↓ cardiac output.

Clinical Picture

Clinically diagnosed by harsh whistling sound of air going through the defect following history of trauma

Symptoms

- History of trauma.
- Acute chest pain, Dyspnea, cough & cyanosis.

Signs

▪ **General:**

- Signs of shock, engorged neck veins, cyanosis & respiratory distress (working ala nasi)

▪ **Local:**

Inspection: - Ecchymosis & bruises
- ↓ chest movements on affected side

Palpation: - Shift of trachea to the opposite side,

Percussion: hyperresonance on the affected side.

Auscultation: ↓ air entry.

- ↓ TVF

Investigations

For diagnosis

▪ CXR:

- Jet-black on affected side.
- Total lung collapse on the affected side.
- Partial lung collapse on the opposite side.
- Tracheal and cardiac shadow shift to the opposite side.
- Flattened diaphragm is displaced downwards on the affected side.

For complications

- ABGs.
- U/S for associated visceral injuries.

Treatment

▪ ABCD at the site of the accident:

- Close the wound by adhesive external dressing.

▪ Resuscitation & monitoring + 1ry survey.

▪ Definitive:

- The wound is sutured with insertion of an intercostal tube under water seal in the 5th space at the MCL.

Tension Pneumothorax

Definition

- Air inside the pleural cavity due to valvular tear which allows air to enter but does not allow it to come out from the pleural space.

Pathology

1. Respiratory failure (impairment of gas exchange due to)

- Marked ↑ in intrapleural pr. → total collapse of the underlying lung.
- Mediastinal shift to the opposite side → partial collapse of the other lung.

2. Circulatory failure due to:

- Loss of the negative intrathoracic pressure → ↓ venous return → ↓ cardiac output.
- Mediastinal shift disturbs heart action.

As open pneumothorax but more severe cardio-respiratory distress

Clinical picture

Symptoms

- History of trauma.
- Acute chest pain, dyspnea, cough & cyanosis.
- Respiratory arrest may occur.

Signs

- General: signs of shock, engorged neck veins, cyanosis & respiratory distress (working ala nasi).

▪ Local:

1. Inspection: -Ecchymosis & bruises.
-↓ chest movements on affected side
2. Palpation: -Shift of trachea to the opposite side. - ↓ TVF
3. Percussion: tympanitic resonance on affected sides.
4. Auscultation: ↓ air entry.

Complications (associated injuries)

- Chest wall: fracture rib and flail chest.
- Pleura: hemothorax.
- Diaphragmatic injury.
- Great vessel injury.
- Cardiac injury.
- Lung lacerations.
- Esophageal injury.

DD***Of acute chest pain + shock***

1. Acute myocardial infarction.
2. Cardiac tamponade.
3. Massive pulmonary embolism.

Investigations***(Rarely needed)***

- Are done if diagnosis is suspicious.
- If not, proceed to urgent treatment that should be done once tension pneumothorax is diagnosed.

Treatment**First aid**

- **Airway:** maintaining upper airways clear, even by insertion of cannula 14G in the cricothyroid membrane.
- **Breathing:** keep normal breathing rate by assisted or mouth to mouth breathing.
- **Circulation:** control of bleeding by compression
- **D** → Damaged limb → should be splinted.
→ Drugs → morphine if needed.

At hospital

1. **Resuscitation & primary survey:**
 - a. Ryle, IV line and catheter.
 - b. IV fluids, blood transfusion, antibiotics & analgesics.
2. **Continuous monitoring of:**
 - a. Level of consciousness.
 - b. Vital signs (pulse, BP, temperature, and R.R).
 - c. CVP.
 - d. Urine output, fluid chart and amount of drained fluid.
3. **Secondary survey:**
 - After establishment of general condition of patient.

Definitive treatment:

- **Wide-bore cannula is inserted** in the 2nd space at the mid-clavicular line then it's replaced by an intercostal tube with under-water seal.

- Continuous bubbling of air through the intercostal tube denotes the possibility of occurrence of broncho-pleural fistula.

Closed Pneumothorax

Definition

- A limited amount of air is entrapped into the pleural cavity d.t lung laceration.
(Not communicating with the exterior)

Symptoms

- History of the cause.
- Chest pain & slight dyspnea.

Signs

- **Inspection:** - ↓ movements at the affected side.
- **Palpation:** - Confirms diminished movements.
- ↓TVF, but there is **NO** mediastinal shift.
- **Percussion:** - Hyper-resonance.
- **Auscultation:** - ↓ air entry.

Investigations

Plain CXR shows

- Absence of lung markings (i.e. Jet-black).
- The edge of the collapsed lung is usually visible.

Treatment

- **If the amount of air is small & there is no dyspnea:**
→ The patient is treated **conservatively** (monitor vital signs & repeated ABGs is done) until spontaneous absorption of the air takes place.
- **If there is dyspnea**
→ An **intercostal chest tube with under water seal** is inserted.
(Until complete expansion of the collapsed lung occurs).

Hemothorax

Definition

- Collection of blood in the pleural space.

Etiology

Local

- 1- Traumatic injury of intercostals vessels, internal mammary vessels or the lung tissue.
- 2- Post-operative.
- 3- Spontaneous due to tumors of lung or pleura.

General

1. Blood disease (hemophilia, purpura).
2. Hypertension.
3. Drugs as anticoagulant.

Pathology

- The respiratory and cardiac movements defibrinate the blood, so that the collection remain fluid in most cases.
- Blood in the pleural space is very irritant exciting the formation of a considerable effusion that is rich in proteins.
- In neglected cases a fibrin layer is deposited on both layers of the pleura, later this deposit is transformed to a fibrous layer. The end result is a collapsed lung and deformity of the chest wall with crowding of the ribs.

- In traumatic cases, there is commonly associated pneumothorax, i.e., hemopneumothorax.

Clinical Picture

Symptoms

- History of trauma.
- Dyspnea, chest pain.
- **Hemoptysis** (if lung laceration occurs).

Signs

- **General:**
 - Signs of shock, engorged neck veins, cyanosis, working ala nasi & associated injuries
- **Local:**
 - **Inspection:** ↓ chest movements
 - **Palpation:** ↓TVF
Tenderness
Shift of trachea to opposite side
 - **Percussion:** **Dullness**
 - **Auscultation:** ↓ air entry.

Fate & complications

(Hemothorax is never to be absorbed spontaneously)

- Motion of the diaphragm, lungs & the intrathoracic structures → defibrination of the blood → incomplete clotting.
- If untreated it undergoes the following changes: (complications)
 - Empyema: due to bacterial contamination of the retained hemothorax & if neglected → bacteremia or septic shock.
 - Fibrothorax (pleural fibrosis): due to fibrin deposition which coats both visceral & parietal pleural surfaces trapping the lungs.

Investigations

For diagnosis

- **CXR** → If the hemothorax is less than 500 ml, it will lead to obliteration of costophrenic angle & if more than 500 ml it will lead to an opacity rising to axilla + site of fracture.
- **Aspiration** → blood.

For complication

- ABGs.
- Investigations for associated injuries.

Treatment

1. **ABCD** (in poly-traumatized patient).
2. **Resuscitation & monitoring then 1ry survey**
3. **Complete removal of the blood from the pleura** by:
 - A. **Repeated aspiration** (if small in amount).
 - B. **Intercostal (I.C.) tube with underwater seal** (if re-accumulating)
 - Why?** To allow full lung expansion (ensured by X-ray).
To prevent clotting, infection, irritation & pleurisy.
4. **Thoracotomy: (indications):**
 - Severe bleeding (200 ml/hour).
 - Persisting bleeding despite conservative measures.
 - Associated intrathoracic injuries.

- Old clotted hemothorax → This is diagnosed by failure of the tube to evacuate the blood.
 - Loculated hemothorax → The tube partially evacuates the blood.
 - Initial volume of blood coming through the intercostals tube > 2L.
5. **For clotting:**
- Fibrinolytics (early).
 - Decortication (if marked).
6. **Deal with associated injuries.**

Fracture Ribs

Etiology

1. **Direct trauma** → May produce fracture at any site. Visceral injury is common as the broken ends are driven inwards
2. **Indirect trauma** → antero-posterior compression of the ribs → fracture at the angle and since the broken ends are driven outwards visceral injury is uncommon.
3. **Muscular violence** → as in convulsions, violent sneezing or coughing.
4. **Pathological fracture.**

Simple & Multiple Fractured Ribs

	Simple	Multiple
Definition	One rib fracture at one point	Multiple ribs fracture each at one point
Sites	▪ Commonest ribs 3rd → 10th	
Treatment	1- Ensure patent airway. 2- Pain relief: ▪ Analgesic up to pethidine. ▪ Intercostal n. block or epidural anaesthesia	
	3- Strapping better avoided	3- Strapping 4- IPPB

Flail Chest (Stove in ribs)

Definition

- More than one rib (at least 3) are fractured at more than one site.
- A segment loses its bony continuity with the rest of the chest wall.

Etiology

- Direct trauma

Pathology

⇒ *The flail part has the following effects:*

1. **The flail part moves paradoxically with respiration**
 - During **inspiration** chest expand while the flail part is **sucked in**
 - During **expiration** chest collapse while the flail part **bulges out**.

2. Respiratory failure (impairment of gas exchange) due to:

Paradoxical respiration; the lung at the side of injury

- **expands** slightly during **expiration**
- **collapses** more during **inspiration**.

Pendulum respiration due to:

- Oscillation of air between the 2 lungs
- The normal lung is always filled with air deficient in O₂ & loaded with CO₂.

3. Circulatory failure due to:

Mediastinal flutter

- The mediastinum move from side to side with respiratory movements leading to kinking of great vessels

Clinical Picture

History of trauma

Symptoms

- Acute chest pain, dyspnea, cough & cyanosis.

Signs

- **General:** signs of shock, engorged neck veins, cyanosis & respiratory distress (working ala nasi)
- **Local:**
 1. Inspection: -Ecchymosis & bruises
 - ↓ Chest movements on affected side
 - Flail segment moves paradoxically with respiration
 2. Palpation: -Tenderness
 - ↓ TVF & shift of trachea to the opposite side if associated with pneumothorax
 3. Auscultation: ↓ air entry.

Complications

- 1- Pneumothorax.
- 2- Hemothorax.
- 3- Rupture kidney.
- 4- Rupture spleen (left side).
- 5- Rupture liver (right side).

Investigations

Investigation of flail chest

1. CXR & CT scan.
2. ABG & KFTs.

Investigation of complications

1. U/S→rupture spleen
2. Hemothorax→obliteration of costophrenic angle + fluid rising to axilla

Treatment

1. First aid: A,B,C,D at the site of accident
2. At hospital:
 - **Resuscitation& monitoring**

- **Definitive treatment:**

- If small flail segment → Control the paradoxical movement by **strapping** the chest over firm pad or fixing it to bedside using towel cl`ips.
- If severe paradoxical respiration → Tracheostomy and **positive pressure respiration** (after exclusion of tension pneumothorax).
- **If there is an indication for thoracotomy** Open reduction & internal fixation (wiring) of fractured ribs

Empyema Thoracis

Definition

- Pus in the pleural cavity

(I) Acute Empyema

Predisposing factors

- More common in children following lobar pneumonia.

Route of infection

- **Direct access** through open wounds,
- **Local spread:**
 - From chest → pneumonia, mediastinitis or osteomyelitis,
 - From abdomen → subphrenic or liver abscesses.
- **Blood spread:** as in septicaemia.

Causative organisms

(Pathological types)

1- Staphylococcal empyema

- D.t. Local spread from pneumonia (the most common) or blood spread from septicaemia.

2- Putrid empyema

- D.t. rupture of lung or subphrenic abscess (mixed infection → E.coli, proteus, pseudomonas).

	3- Pneumococcal empyema	4- Streptococcal empyema
Incidence	More common	Less common
Onset	After lobar pneumonia = metapneumonic empyema	Synchronously with broncho-pneumonia = synpneumonic empyema.
Pus	Thick, green	Thin, yellow
Localization	Adhesions occur early	No adhesions, no localization and empyema is massive.

Stages

- Diffuse stage.
- Localized stage.
- Neglected stage.

Clinical picture**Symptoms**

- **General:** marked constitutional symptoms (FAHM, rigors)
- **Local:** cough, dyspnea & dull aching chest pain

Signs

- **General:** fever, tachycardia, cyanosis & working ala nasi
- **Local**
 - Inspection: ↓ chest movements.
 - Palpation: ↓ TVF + shift of the mediastinum to opposite side.
 - Percussion: dullness.
 - Auscultation: ↓ air entry.

Complications

- **General:** toxemia, bacteremia, septicemia, pyemia.
- **Local:** chronic empyema & spread (→ lung abscess)

Investigations

- **CXR:** obliterated costophrenic angle with opacity rising towards the axilla.
- **Blood picture:** leucocytosis + ↑ ESR.
- **Thoracocentesis:** reveals pus and for C & S.

Treatment**General**

- Rest.
- Analgesics, antibiotics, antipyretics (AAA).

Local

- 1) **Repeated aspiration:** in early stages
Through the 7th space at the midaxillary line with injection of antibiotics (500,000 units penicillin).
- 2) **Closed drainage:** if pus collects rapidly or if pus becomes too thick to be aspirated.
By insertion of intercostal tube connected to an under water seal through 7th space at the midaxillary line.
- 3) **Open surgical drainage:** by rib resection + surgical evacuation of pus.
Only when full localization has occurred.

(II) Chronic Empyema**Definition**

- Empyema in which the lung is not able to expand after evacuation of pus due to fibrosis.

Types**(1) Open chronic empyema (chronic chest wall sinus):**

It is due to inadequate treatment of acute empyema

- Inadequate drainage (too early, too late, too high or too low drainage).
- Inadequate post-operative care: as early removal of the IC tube.
- Underlying disease: in chest wall (osteomyelitis of ribs), pleura (TB, FB) lung (abscess or tumors).
- Poor general condition: anemia, D.M. ... etc.

(2) Closed chronic empyema:

- Encysted empyema: develops insidiously as the closed collection of pus is completely walled off by adhesions.
- Recurrent empyema: discharging pus intermittently into a bronchus through a bronchopleural fistula.

Clinical picture**General**

- Chronic toxemia with clubbing.
- Acute exacerbations with fever & chills.

Local

- Open type → chronic sinus in the chest wall leading to the pleura and discharging pus.
- Closed type → **Empyema necessitans**: which perforate an intercostal space leading to a subcutaneous abscess (gives an expansile impulse on cough)
- Due to fibrosis:
 - Contracture and rigidity of chest wall with crowding of ribs from fibrosis.
 - Elevation of the diaphragm.
 - Shift of the mediastinum towards the affected side due to fibrosis.
 - Scoliotic deformity of the spine.

Complications

- A. **General**: septicemia, pyemia, 2ry amyloidosis in kidney.
- B. **Local**: spread of infection to the surroundings e.g.
 1. Pulmonary fibrosis.
 2. Bronchopleural fistula.
 3. Sinus.
 4. Empyema necessitans.
 5. Subphrenic abscess.

Investigations**Laboratory**

- Culture and sensitivity: for sputum or pus.
- Complete blood picture: anemia, ↑ E.S.R + leucocytosis.

Radiological

- Plain CXR:
 - Overcrowded ribs
 - Shift of trachea
 - Elevation of diaphragm
 - May show the underlying cause
- Lipidol pleurogram (obsolete): shows the size of the cavity + any bronchial communications.
- CT scan chest & biopsy: most accurate.

Instrumental

- Bronchoscopy to detect any cause.

Treatment**General**

- Correction of anemia & control DM
- Antibiotics according to culture & sensitivity.
- Multivitamins.

Local

- **Re-drainage** by rib resection in a dependent position + proper physiotherapy to allow lung expansion and obliteration of the cavity.
- If expansion of the lung doesn't follow redrainage :
 - **Decortication**: the thick visceral pleura is excised and the lung is expanded by positive pressure ventilation.
 - **Thoracoplasty**: for localized cases, the cavity is obliterated by allowing the chest wall to adhere to the lung.
- If there is underlying localized lung pathology like TB → **pleuro-lobectomy** or **pleuro-pneumonectomy** is done.

Cardiac arrest & Cardio-Pulmonary Resuscitation (CPR)

Definition

- Sudden failure of the heart to maintain circulation (NO COP).

The brain can tolerate no more than 3 minutes of arrest before irreversible damage and death.

Types

(Differentiated by ECG)

- 1- Ventricular fibrillation (VF) → the most common type and the best survival rate.
- 2- Asystole.
- 3- Electromechanical dissociation.

Causes**A- Myocardial depression:**

- 1- Myocardial anoxia e.g. myocardial infarction, shock, etc...
- 2- Metabolic:
 - a- Hyerkalemia / hypokalemia.
 - b- Hyponatremia.
 - c-Acidosis.
- 3-Drugs e.g. adrenaline IV → VF
- 4-Vagal stimulation e.g catheterization.

B- Inadequate venous return:

- 1- Mechanical:
 - Massive pulmonary embolism.
 - Pleural effusion.
 - Cardiac tamponade.
- 2- Acute hemorrhage.
- 3- General or spinal anesthesia.

Diagnosis

- 1- Sudden loss of consciousness.
- 2- Absence of carotid pulse.
- 3- Cessation of respiration.
- 4- Bilateral dilated fixed pupil (relatively late sign)

Management

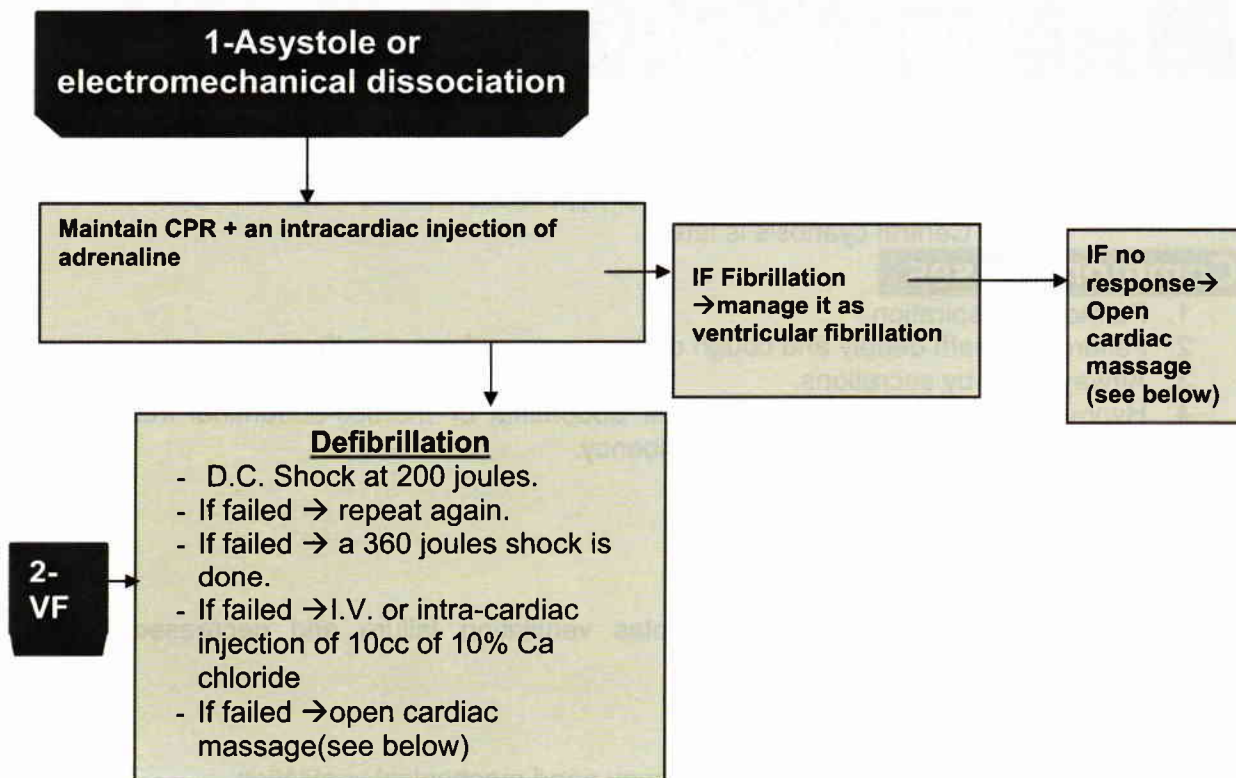
- The proper management is carried out immediately and consists of three phases.

1. Pre-hospital management (cardiopulmonary resuscitation; CPR)

- External CPR by direct mouth-to-mouth breathing and closed cardiac massage.
- The aim is not to restart the heart but to provide an artificial circulation and artificial ventilation until appropriate facilities are available.
- **Technique:**
 1. The patient is laid on firm surface and the legs raised to increase the V.R. the head is tilted back with one hand to ensure clear airway, and the nostrils are closed by the other hand while the mouth is applied to the patient mouth's to fill his lung with expired air at a rate of 10-15 times per minute.
 2. At the same time, another person performs external cardiac massage by compressing the lower half of the sternum against the spine at a rate of about 100 times per minute.

2. Hospital management:

- a. The aim is to restore the normal cardiac rhythm.
- b. An endotracheal tube is passed and the lung are ventilated with O₂.
- c. A venous cannula is inserted and suitable infusions are given.
- d. ECG monitoring is done to differentiate cardiac asystole from ventricular fibrillation.



➤ **OPEN CARDIAC MASSAGE :**

- The chest is opened through the left 5th intercostal space and the pericardium is widely opened to permit bimanual massage with one hand above and the other below the ventricular mass.
- Massage is continued at a rate of 60-80/min until the cardiac tone returns and the heart becomes smaller, firmer and pinker.

3. **Subsequent treatment (in ICU):**

(After correction of normal rhythm)

- Control blood pressure (kept over 90)
 - If the chest has been opened it is left open for half an hour to watch for refailure of the heart.
 - Control hyperkalemia → CaCl_2 , glucose, insulin.
 - Control hypothermia & dehydration.
 - Control metabolic acidosis → NaHCO_3 8.4%
 - If recurrent VF → prophylaxis lignocaine or implantable cardioverter defibrillator.
 - Anticonvulsants agents for convulsions (may result from severe anoxic cerebral damage).
- ⇒ After stabilization of the case, find and treat the primary cause (e.g. coronary bypass surgery for coronary occlusion).

Post-operative Hypoxia

- ▶ It is common and serious condition. It manifests clinically by
 - Restlessness, anxiety or confusion.
 - Tachpnea.
 - Tachycardia, arrhythmias or hypotension.
 - Central cyanosis is late.

Common causes

1. Pulmonary aspiration.
2. Failure to breath deeply and cough during recovery from anesthesia.
3. Airway block by secretions.
4. Hypoventilation due to pain of upper abdominal or thoraco-abdominal incisions, opiates overdose or prolonged recumbency.
5. Pulmonary embolism.
6. Pulmonary edema.

Investigations

1. Pulse oximetry.
2. ABG; increased PCO_2 denotes ventilation failure and decreased PO_2 denotes oxygenation failure.
3. Chest x-ray

Treatment

- Treat the specific cause. The patient may need mechanical ventilation.

8

PLASTIC SURGERY



Burn

Definition

- Necrosis of tissues by physical or chemical agents.

Etiology

A-Physical Burns

- Thermal (75% of all burns):e.g.boiled water, flame burn**
- Electrical burns.**
- Radiation burns.**

B- Chemical burns

Due to alkalis or acids.

Classification of burns (assessment of burns)

➤ According to the extent:

- A major burn
 - More than 30 % of the body surface area.
- An intermediate burn
 - 15 - 30% in adults.
 - 10 - 30% in children.
- A minor burn
 - Less than 15 % in adults.
 - Less than 10 % in children.

▪ **This is estimated by:**

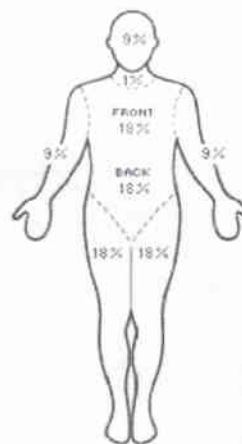
Rule of 9:

➤ According to the depth:

1st degree: affect epidermis, heals rapidly, **app.:** blisters surrounded by erythema, moist, painful and sensitive to air.

2nd degree: affect epidermis and portion of dermis, late healing, **app.:** blisters surrounded by erythema, moist. painful and sensitive to air.

- **3rd degree:** complete destruction of dermis&epidermis, no healing, **app.:** white or black scars, dry, painless.



Pathophysiology (pathological sequale)

- 1- Increased capillary permeability in burnt area.
- 2- Excessive loss of water by evaporation through burnt skin → severe dehydration and catabolism,
- 3- Thermal injury to the skin results in 3 zones:
 1. **The central inner zone (zone of coagulation):**
 2. **The intermediate zone (zone of stasis):**
 3. **The outer zone (zone of hyperaemia):**

Complications

The primary cause of death in burnt patients is infection.

a- Systemic:

1- Inhalation injury:

- a- Immediately: Asphyxia.
- b- Later:
 - Bronchospasm, atelectasis, pneumonia, pulmonary embolism and pulmonary edema may follow.
- c- Finally: type II respiratory failure with systemic sepsis.

2- Shock may be:

- a- Neurogenic(immediately): d.t severe pain.
- b- Hypovolemic(early): d.t. ↓ plasma vol.
- c- Septic(late): d.t. infection.

3- Renal complication:

Oliguria, fluid and electrolyte imbalance and acute tubular necrosis → acute renal failure.

4- Cardio-vascular complication:

Decrease cardiac output then H.F., anemia, hypoproteinemia, hypofibrinogenemia and increased blood viscosity → D.V.T.&P.E.

5- Gastro-intestinal complications:

- a- Stomach → acute gastric dilatation.
- b- Stomach and duodenum → curling ulcer.
- c- Intestine:
 - Ileus and acute ulceration of colon secondary to fungal infection.
- d- Liver: hepatic dysfunction.

6- Endocrine system complications:

- ↑ Catecholamines from stress, ↑ Cortisol and ADH and ↑ Protein catabolism.

7- Multiorgan failure.

8- Psychological complications: e.g. anxiety, irritability, depression.....

b- Local complications:

I. Early local complications:

1. Infection:

- It occurs usually between 4 – 7 days post-burn.
- Treatment by proper local burn wound care (discuss).

2. Constricting eschars:

- The limbs → ischemia or the chest → dyspnea.

3. Suffocation:

- Due to edema in burns of the face and neck.

4. Compartmental syndrome: due to edema of the subcutaneous tissue.

II. Delayed local complications:

1. Contractures: across joints.

2. Scar formation: (hypertrophic or keloid)

3. Malignant transformation: (Marjolin's ulcer) in long-standing unstable scars is rare.

Diagnosis of Burn

1- C/P of burn:

- 1- Pain: immediate and intense in superficial burns but little pain in deep burns.
- 2- Swellings: local tissue edema.
- 3- Disturbance of function: complications.

2- Diagnosis of type of burn:

	Partial thickness burns	Full thickness burns
Appearance	- Forms blisters surrounded by erythema. - Their surface is moist due to exudation of plasma.	- White or black eschars - The area is dry - Possible visible thrombosed S.C vessels
Presence of pain	- Painful - Sensitive to air	Painless (due to affection of nerve ending)
Epilation test	Hair comes out with difficulty	Hair comes out easily
Rate of healing	- Heal within 3 weeks - Usually heal without scar	- Granulation tissue formation and eschar separation starts to occur after 3 weeks - Heals with scar formation

3- Diagnosis of severity of burn (prognosis)

Extent of burn, depth of burn, site of burn, infection, type of burn, associated injuries., Age of patient and Concomitant diseases:.

Treatment of Burn

A-First aid treatment

- **Move** the patient from the source of burn and remove the patient's clothes.
- **Airway**: maintained
- **Breathing**: maintained.
- **Circulation**: maintained.
- **Drugs**: IV analgesic up to morphia, antitetanic Immunoglobulins
- **Exposure**: remove any clothes.
- **Run water**: at room temperature.

B-Definitive treatment

1- For minor burns:

Can be treated as out patients by:

- **Clean** with antiseptics solution as Savlon® or Betadine®.
- **Dressing**: repeated for 2 weeks by silver sulfadiazine+Vaseline gauze.
- **Give** analgesic and prophylactic systemic antibiotic.

2- For major burns:**a- General treatment:**

- **Admission of the patient to burn unit of hospital.**

- **Resuscitation and monitoring:**

- Wide bore IV cannula is inserted.
- A Foley's urethral catheter.
- Ryle tube suction and nothing by mouth in the first 48 hours then oral feeding should be started gradually after that.
- Central venous line may be needed:
- Monitoring of:
 - o Vital signs, urine output, cvp, consciousness level.

- **Fluid therapy:**

- 1- **Timing:** It is essentially given for the first 48 hours and after that according to the response of patient.

- 2- **Amount and rate:**

- Amount infused during the first 48 hours is 2 ml/percent surface area burn/kg body weight.
- The amount and rate of fluid replacement depends on:
 - Patient's body weight.
 - Percentage of total body surface area injured.

- 3- **Types of fluids:**

- Only crystalloids (saline or lactated ringer) over the 1st day, others prefer half the amount colloids and the other half crystalloids.

- 4- **Formulae and fluids in common use: (2 Formulae)**

- **Evan's formula:**

- 1st day: 1 ml/kg/% burn normal saline + 1 ml/kg/% burn colloid + 2000 ml glucose.
- 2nd day: 0.5 ml/kg/% burn normal saline + 0.5 ml/kg/% burn colloid + 2000 ml glucose.

- **Parkland formula(the commonly used):**

- 1st day: 4 ml/kg/% burn lactated ringer's + 2000 ml glucose.
- 2nd day: 1ml/kg/ % burn lactated ringer's + 1ml/kg/% burn colloid + 2000 ml glucose.

- 5- **Monitoring of adequacy of intravenous resuscitation:** by vital signs and urine output.

b- Local burn wound care**The aim is to avoid infection****A- Early care:** after general resuscitative measures, attention should be directed to burn wound.

- 1- Escharotomy: in constricting eschars of the limb or chest.
- 2- Fasciotomy: in deeper burns.
- 3- Cleaning and removing loose skin and tissue debridement.
- 4- Topical antimicrobial agent:
- 5- After application of local cream, the wound is managed by either exposure method or by occlusive method.

	Exposure method	Occlusive method
Definition	The wound is managed by leaving it exposed under complete aseptic precautions	The wound is covered by bulky occlusive dressing changed every 2-3 days
Indications	1- Burns of face, neck, perineum 2- Burns involving one side of trunk	Circumferential burns of Limbs or chest .
Advantages	1- More comfortable to patients. 2- Avoid repeated dressing. 3- Decrease anaerobic infection.	1- Prevent cross infection. 2- ↓ fluid loss by evaporation. 3- ↓ pain by covering exposed nerves. 4- ↓ Edema of tissue by compression

B- Later care for deep wounds

- 1- Escharotomy.
- 2- Synthetic grafting as silicone or semi-synthetic grafting.
- 3- Autologous skin grafting.
- 4- Biological grafting: when autografts are not enough e.g. allograft or xeno graft.
- 5- Prevention and treatment of late complications e.g. contracture and deformity by proper splintage and immobilization in the best function position and physiotherapy.

Skin Grafts & Flaps

1- Skin Grafts

Definition

- It is segment of epidermis & variable thickness of dermis that has been completely departed (cut) from its blood supply & donor site → transferred into a recipient site (skin loss)

Types

- Split thickness graft (**Thiersch graft**)
- Full thickness graft (**Wolf graft**)

Steps

- Separate of required segment by surgical knife (dermatome)
- The recipient area should be surgically clear & has formed granulation tissue (capable to regenerate due to adequate blood supply)
- After application of the graft:** The process of **(TAKE)** takes place which aims to nutrition of the graft by
 - Imbibitions of plasma (48 hrs) from newly blood vessels
 - Vascularization (after wards)
- Healing of donor site :** depends on degree of thickness Split or full.

	SPLIT THICKNESS	FULL THICKNESS
Definition	Epidermis + superficial part of dermis	Epidermis + full thickness of dermis
Indications	- Covering wide area of granulating T. - Coverage after Deep burn. - Malignant tumor resection.	- Facial wounds - Palm of hands and sole of feet.
Donor sites	- Trunk , thighs - Upper arm, fore arm	- Post-auricular skin - Upper eye lid, supraclavicular
Advantages	- Easy separation & application - Increase TAKE by graft. - Can cover wide area. - Early detection of recurrence of malignancy - Donor site heals spontaneously.	- Direct closure of donar site. - Less liable to contraction & pigmentation. - Better sensation - Better cosmesis - Strong resistance to trauma.
Disadvantages	- More liable contraction. - More liable for pigmentation. - Weak resistance to trauma. - Poor sensation & cosmesis	- Difficult separation & application. - Less TAKE by graft. - Limited donor site. - Late detection of recurrence of malignancy. - Scar at the donor site. - Can't be applied on granulation tissue

2- Flaps

Definition

- Tissue transferred from an area of the body to another area but retaining attachment to its original site for its blood supply for nourishment.

Indications

- Wound with proper vascular bed e.g. overlying bare bone.
- In weight bearing area e.g. sole.
- Reconstruction of facial features.

Types

1- Skin flaps

A- Local skin flaps:

1- Random pattern:

- Have no anatomically recognized blood supply so it has limited length(1:1) and limited area of rotation.

2- Axial pattern:

- Have anatomically known blood supply so no limitation in length to width ratio.
- Axial pattern flaps are subdivided into 2 types:
 - a- Axial pedicle: it retains its attachment to donor area.
 - b- Island flap: it retains its blood supply without skin attachment.

B- Distant flaps:

- ▶ Distant pedicle flap: they keep the recipient site attached to the donor site for a period of 2-3 weeks in order to allow for neovascularization before separating the base e.g. cross leg flaps

2-Myocutaneous flaps

- These are axial flaps that receive their blood supply from the underlying muscle through perforator vessels.
- The muscle is lifted from its bed with overlying fascia and an area of skin then the whole flap is rotated in an arc to cover the raw area.
- Latissimus dorsi myocutaneous flap used to cover the anterior chest wall after radical mastectomy.

3- Fasciocutaneous flaps

- To avoid functional deficit of muscle transfer and a bulky flap they have the same principles of myocutaneous flaps but their blood supply is through fasciocutaneous perforators

4- Microvascular free flaps

- It is now possible to anastomose vessels of less than 1 mm in diameter.

Skin Neoplasm

Basal Cell Carcinoma (Rodent ulcer)

Definition

- **Locally malignant** tumor from basal layer of the epidermis or from equivalent cells of hair follicles, sweat or sebaceous glands

Incidence

- Age: > 40 years
- Sex: Male > Female

Predisposing factors

- Exposure to sun rays, Ultra-violet rays (**most important**) so the disease is more common in Farmers & Sailors.
- Albinism & Xeroderma Pigmentosa (AR).
- Ionizing radiation.
- Immunocompromized Patients.

Pathology

Site

- **90% in the face in Area** above a line from lobule of the ear till angel of mouth (Mostly at inner canthus of the eye & nasolabial fold)

Gross picture

A- Common types:

- 1- **Nodular form**: pearly white or blue translucent nodule with dilated capillaries over it (it is the earliest lesion).
- 2- **Ulcer form** (Rodent ulcer) → **the commonest**
 2. **Shape** → rounded, oval or irregular.
 3. **Number** → usually single but may be multiple
 4. **Base** → indurated, early mobile & late fixed.
 5. **Floor** → necrotic
 6. **Edge** → rolled in, beaded
 7. **Magin** → early soft & late indurated.
 8. **L.N.** → not enlarged (locally malignant)
(If enlarged → infection or SCC change)
 9. **Discharge** → blood or pus.
- 3- **Excavating type**: the ulcer erodes deeply into the underlying structures leading to destruction of the nose and infiltration of the nasal sinuses.

B- Uncommon types

- A. Pigmented type (DD melanoma).
- B. Cystic type.
- C. Flat superficial spreading BCC
- D. Cicatricial type.
- E. Sclerosing type.

Microscopic

- Rounded solid masses of cells (pallisade appearance).
 - Low columnar epithelium at periphery as (basal cell).
 - Polyhedral cells at center as (Prickle 's cells)
- The masses are separated by fibrous tissue stroma.

Spread

- Only direct spread to surrounding (locally malignant)

Complications

- Infiltration of the surrounding (it may erode the underline skin)
- Secondary infection → meningitis and cavernous sinus thrombosis (Cause of death)
- Hemorrhage.
- Epitheliomatous transformation (Baso-squamous carcinoma)

Clinical picture

- Type of patient:
 - ♂ > 40 years
 - Fair colored people
 - Farmers & sailors
- Nodule: Painless nodule → Ulcer resistant for treatment
- Ulcer:
 - Slowly growing but progressive.
 - Characters : as before (in pathology)
- Lymph Nodes: → Not enlarged except in:
 - 2ry infection → firm, tender and mobile.
 - Epitheliomatous transformation → stony hard, mobile but later fixed.

Epitheliomatous transformation is suspected if:

- 1) Growth becomes rapid
- 2) Edge become everted
- 3) LN becomes hard & fixed

Differential Diagnosis

1. Ulcer of Squamous Cell Carcinoma (SCC).
2. Malignant melanoma.
3. Cock's peculiar tumor.
4. Septic granuloma.
5. Chancre.

Investigations

It's mainly a clinical diagnosis but biopsy is mandatory to ensure diagnosis & exclude D.D.

1. Biopsy (Excisional or incisional):
2. X-ray → To detect bony involvement(rodent ulcer).

Treatment

- 1- Surgical(main line): excision with 1 cm safety margin & skin graft or 1ry closure.
- 2- Irradiation: indicated if pt. is unfit or refusing surgery.

Prognosis

- **Good prognosis as it is a locally malignant tumour.**

Squamous Cell Carcinoma

Age & sex

- Male > 40 yrs

Predisposing factors

- Exposure to sun rays, Ultra-violet rays (**Most important**) so the disease is more common in Farmers & Sailors.
- Previous Irradiation.
- Albinism & Xeroderma Pigmentosa (AR).
- Long standing irritation of the skin as in chronic ulcers, old burn scar & Marjolin's ulcer.
- Prolonged exposure of the skin to carcinogenic agents as coal tar derivatives.
- Immunocompromised Patients.

Pathology

- Site**
 - Any site covered with Sq. epith especially upper part of face, lower lip and the dorsum of hands.

Gross pic.

- Types**
 - Nodular form: earliest lesion
- Ulcer (CCC): (commonest)**
 - Shape** → Rounded, oval or irregular.
 - Number** → usually single but may be multiple
 - Base** → Indurated, early mobile late fixed.
 - Floor** → necrotic
 - Edge** → everted
 - Margin** → early soft & late indurated.
 - L.N.** → Early enlarged & soft (due to infection)
Late hard (due to infiltration)

Microscopic

- Differentiated SCC → with cell nests (Prickle cells + keratin in the center)
- Undifferentiating SCC → with masses of malignant cells (Anaplastic cells with basophilic cytoplasm which provide no cytological evidence of their origin)

Spread

- Direct & lymphatic, rarely blood

N.B: in a patient with Marjolin's ulcer the scarring makes lymphatic spread late.

Complications

- Infiltration of the surrounding
- Secondary infection
- Hemorrhage
- distant metastasis & cachexia

Clinical picture

- Nodule: Painless nodule → Ulcer resistant for ttt.
- Ulcer
 - Rapidly growing
 - Characters : As before (in pathology)
- Lymph Nodes
 - Enlarged in
 - 2ry infection → elastic & tender
 - Metastasis → Hard & fixed

Differential Diagnosis

1. Rodent ulcer
2. Malignant melanoma.
3. Septic granuloma.
4. Chancre
5. Keratoacanthoma.

Investigations

1. Biopsy → excisional or incisional
2. X-ray → To detect bony involvement
3. Sentinel L.N study: affected L.Ns
4. C.T. scan

Treatment**A -Surgical (main line)**

1. Excision with safety margin 2 cm except in face where is 1cm & Skin graft or by Primary closure

B - Irradiation

Indications → patients unfit or refusing surgery.

Contraindications

1. Radio-resistant lesion.
2. Ulcer infiltrate bone and cartilage (to avoid irradiation necrosis).
3. Ulcer near the eye (to avoid irradiation Cataract).
4. Recurrence after irradiation (to avoid irradiation resistance).
5. Marjolin's ulcer.

If LNs enlarged by metastasis (SCC only)

1. If LNs are close to tumor → excised with 1ry lesion in 1 block.
2. If LNs are away from tumor → block dissection later on (after 2 wks), to avoid postoperative lymphedema and picking up of micrometastasis.
3. No prophylactic block dissection in skin malignancy.

Prognosis

- Depends on patient's age, differentiation & L.Ns affection

Benign Melanoma (Naevus)

Definition

- True neoplasm arising from melanocytes.

Types

1. Lentigo

- Melanocytes replace the basal layer of epidermis in certain sites.
- **Histologically:** appear as rounded cells containing fine granular melanin pigment.
- **Clinically:** flat spot black or brown in color

2. Junctional pigmented naevus

- **Histologically:**
 - More proliferation of melanocytes → small nodules of epidermis that bulge downward in the dermis.
- **Clinically:** like lentigo

3. Compound naevus

- **Histologically:**
 - The basement membrane is disrupted and some melanocytes pass to the dermis, these melanocytes lose their capacity to melanin and called naevus cells also the dermis contains some macrophages containing melanin pigment.
- **Clinically**
 - Black or brown nodule raised above surface of skin with some hair sometimes, it occupies large area → giant hairy naevus.

4. Intradermal

- **Histologically:**
 - Melanocytes are present mainly in the dermis with junctional activity in majority of cases.
- **Clinically:** like compound naevus.
 - **Indications for surgical excision:**
 - 1- Cosmetic reasons
 - 2- If subjected to repeated trauma.
 - 3- If suspicion of malignant transformation.
 - **Can pigmented naevus turn malignant?**
 - 1- Giant hairy pigmented naevi.
 - 2- Junctional naevus.
 - 3- If a site of repeated chronic irritation e.g. during shaving.

Malignant Melanoma

- It is either denovo or on top of benign melanoma.

Q: What are the criteria of malignant transformation of benign naevus?

- 1- ↑ Size and thickness.
- 2- ↑ Pigmentation.
- 3- Occurrence of itching, tingling, ulceration or bleeding.
- 4- Development of satellite nodules around it.
- 5- Hard in consistency or enlarged hard draining LNs.

Predisposing factors

- 1- Prolonged exposure to sunlight (UV rays)
- 2- On top of benign melanoma.
- 3- Albinism, retinitis pigmentosa.
- 4- Racial factors (rare in negros).

Incidence

- Disease of adult and old age.
- Male > female.

Pathology and clinical types (according to WHO classification)

1- Superficial spreading melanoma (the most common) 64%

- **Age:** usually middle age.
- **Site of lesion:** any part of the body
- **Character of lesion:** raised above surface and has irregular edge.
- **Prognosis:** good as malignant melanocytes spread along the epidermis (radial growth).

2- Nodular melanoma 12-25%

- **Age:** young.
- **Site:** any part of body.
- **Character:** raised above surface, grey or black in color with smooth surface and liable for ulceration.
- **Prognosis:** bad as malignant melanocytes invade the dermis (vertical growth)

3- Lentigo maligna (Hutchinson's melanotic freckle) 1-15%

- **Age:** elderly person
- **Site:** usually in face
- **Character:** it begins as a flat brown macule which grows very slowly.
 - Some areas may regress.
- **Prognosis:** it is the least malignant type.

4- Acral lentiginous melanoma

- **Age:** middle-age.
- **Site:** palm, sole, under nail
- **Character:** may simulate any other type.
- **Prognosis:** very poor (starts by radial then later on vertical growth)

5- Amelanotic melanoma

- **Malignant melanoma** which arise in the eyes, meninges or at mucocutaneous junctions as the anal canal.
- **Diagnosed by:** DOPA reaction test.
- **Prognosis:** poor.

Spread

- **Direct** → radial or vertical growth.
- **Lymphatic** → deposits & retrograde permeation with satellite nodules around the tumour.
- **Blood** → high affinity to liver metastasis (especially from choroids).

Staging (Clark's histological stages)

I- Clark's classification (according to depth in skin):

- Epidermal.
- Dermo-epidermal junction.
- Superficial papillary dermis.
- Deep papillary dermis.
- Subcutaneous tissue.

II- Breslow classification ✓ (more used)

1. Skin infiltration **< 0.75** mm.
2. Skin infiltration **0.75 – 1.5** mm.
3. Skin infiltration **1.5 - 3** mm.
4. Skin infiltration **> 3** mm.

Diagnosis

A-Clinical picture:

1. C/P of malignant melanoma

(The only sure method of diagnosis is histological examination, however malignant melanoma can be suspected clinically with A, B, Cs of melanoma)

► Symptoms

- 1- **Denovo lesion.**
- 2- **Malignant changes** in benign naevus.
→ Rapidly growing, itching, pain, change of color, hemorrhage or ulcer.
- 3- Occult presentation.
- 4- **Transit metastasis** → Enlarged micro-metastasis after removing of the 1st tumor.

► Signs

1. **Nodule or ulcer** of variable colors (from amelanotic to black)
2. **Satellite lesions** may be seen.
3. **Regional L.N. or liver metastasis**

► Malignancy is suspected with A, B, Cs of Melanoma

- 1- **Asymmetry of shape:** one half does not look like the other.
- 2- **Border** is irregular, poorly defined, and discontinuous.

- 3- Color is variable: varied from one area to another, shades of tan, brown, black and sometimes white, red or blue.
- 4- Diameter is larger than 6mm in most cases (diameter of pencil eraser).
- 5- Enlargement: gradual increase in size and **Elevation**.

2. Clinical types (See before)

3. DD

- | | |
|------------------------------------|-----------------------------------|
| 1- Granuloma. | 3- Hemangioma |
| 2- Pigmented basal cell carcinoma. | 4- Compound or junctional naevus. |

B- Investigations

- 1- To confirm diagnosis → biopsy
- 2- For staging → isotope scintigraphy for sentinel LNs
- 3- Routine preoperative investigations.

Treatment

A- Operable:

2- For the 1ry tumor:

- Surgical excision with adequate safety margin of skin and subcutaneous tissue but not including the deep fascia + plastic reconstruction:
 - If tumor thickness < 1 mm → safety margin is 1 cm.
 - If tumor thickness 1-4 mm → safety margin is 2 cm.
 - If tumor thickness > 4 mm → safety margin is 3 cm.

3- For draining LNs:

- Radical dissection is performed if they are large and firm but if not frankly malignant by clinical examination → fine needle aspiration cytology is performed.

B- Inoperable tumor

- Metastasis is treated by interferons, chemotherapy and interleukin-2.

Prognosis

- According to type, rate & depth of growth.

Congenital anomalies of the face

1. Cleft palate.
2. Preauricular sinus. This is due to imperfect fusion of the auricular tubercles.
3. Dermoid cysts. Sequestration dermoid cysts occur at the lines of embryonic fusion. The most frequent is the external angular dermoid.
4. Pierre Robin syndrome. Consists of cleft palate associated with receding mandible and posterior displacement of the tongue obstructing the oropharyngeal airway.
5. Mandibular prognathism

Cleft Lip (Hare Lip) & Cleft palate

Incidence

- The most common congenital anomalies of the orofacial structures.
- Typical distribution of cleft types is:
 1. Cleft lip alone 15%.
 2. Cleft lip and palate 45% (more in males).
 3. Cleft palate alone 40% (more in females).

Etiology

- Genetic predisposition:
 - Family history in a 1st degree relative ↑ the risk to 1 in 25 live births
- Environmental factors:
 - Drugs: steroids, diazepam and phenytoin.
 - Irradiation.
 - Viral infections (e.g rubella)

Cleft Lip (Hare Lip)

Definition

- Failure of fusion between the median part of frontonasal process and one or both lateral maxillary processes in developing face.

Types

I) Lateral or median:

- **In the upper lip**: usually lateral.
- **In the lower lip**: it is median.

II) Unilateral or bilateral:

- Unilateral in 85% of cases. More in the left side
- Bilateral in 15% of cases

III) Complete or incomplete:

- **Complete** → The cleft extend upward to the nostril.
- **Incomplete** → The cleft does not extend to the nostril.

IV) Complicated or uncomplicated:

- **Complicated** → With cleft palate, the commonest.
- **Uncomplicated** → Without the cleft palate.

Diagnosis

Antenatal diagnosis:

By U/S scan after 18 weeks of gestation (if antenatal diagnosis is confirmed, referral to a cleft surgeon for counseling to allay fears) (reassure the patient)

Clinical picture & Complication:

1. Flaring & flatness of nares on affected side if complete.
2. Disfigurement.
3. Abnormal Teeth Growth
4. Nasal tone.
5. Short lip-nose distance.
6. In 35% of cases → Associated congenital anomalies
e.g. cleft palate, coloboma, encephalocele etc.

Treatment

- Feeding of the baby using spoon
- Surgical: Millard's operation at the age of 3-6 months, infant should be 10 pounds in weight, Hb at least 10 mg%.

Cleft Palate

Types

1. Cleft uvula.
2. Cleft soft palate.
3. Cleft soft and hard palate (complete)
4. Complete cleft + one side of premaxilla (Bipartite cleft).
5. Complete cleft + both sides of premaxilla (Tripartite cleft).

Diagnosis

Antenatal diagnosis:

- All types (except isolated cleft palate) can be diagnosed by U/S scan after 18 weeks of gestation (then referral to a cleft surgeon)

Clinical picture & Complication: (Due to communication between the oral & nasal cavities):

- Impairment of suckling: due to inability to create -ve intra-oral pressure.
- Impaired speech: due to inadequate retropharyngeal mechanism + nasal tone + hearing loss).
- Impairment of dentation if the alveolar margin is reached.
- Impaired sinus, otitis media due to nasal regurgitation due to impaired aeration of Eustachian tube.
- Recurrent chest infection (due to reflux into nose).

Treatment

- **Time:** 12-18 months

Pre-operative management:

- 1- Attention to feeding. As there is inefficient breast feeding, a bottle with a large hole is used or spoon feeding in an upright position.
- 2- Prevention and treatment of chest infection.

▪ **Objectives of surgery:**

- Closure of oro-nasal communication.
- Achieving a competent velopharyngeal sphincter.

▪ **Principles of surgery:**

- paring of edges.
- suturing is done in three layers in the middle line (nasal mucosa, muscle layer, then oral mucosa).
- lateral relaxation incisions are needed.
- fracture of the pterygoid hamulus to relax tensor palati

▪ **post-operative treatment:**

- speech therapy.
- orthodontic treatment.

Swelling of the Jaw

Odontomes

- They are tumors arising from embryonic remnants of teeth.
- The most important are the epithelial odontomes.

Epithelial odontomes

1-Dental Cyst	2-Dentigerous Cyst
- It doesn't contain a tooth	- It contain a tooth
- More common in upper jaw	- More common in the lower jaw
- Usually about 40 years	- It occurs in children

Treatment: excision & bone graft or curettage & bone graft.

3-Adamantinoma (Ameloblastoma)

- Clinically it is an epithelial tumor which arises probably from ameloblasts i.e. the enamel - forming cells.

Clinical Picture:

Age & sex → It is more common in female & usually occurs in between 20 & 30 years.

C/O → Painless progressive swelling of the angle of the mandible.

- It causes expansion of the jaw, mainly external .
- There may be egg - shell crackling.

X-Ray → (soap - bubble appearance).

Treatment:

- Excision of the affected part of the mandible followed by bone grafting.

Tumors of the Gum (Epulis)

Epulis is a mass of the gum.

1- Fibrous epulis

- It is the commonest type.
- It is a fibroma which rises from the periosteum.
- It is more common in the mandible between the incisors.

► **Clinical Picture:**

- It gives slowly growing, painless, firm swelling which may be sessile or pedunculated.
- It is covered by intact mucous membrane.
- It may turn fibrosarcoma.

► **Treatment:**

- The tooth or teeth related to the tumor are extracted & wedge of the bone including the tumor.

2- Myeloid epulis

- It is a giant cell bone tumor.
- It is common in the mandible.

► **Clinical Picture**

- It grows more rapidly than a fibrous epulis.
- Well-defined sessile swelling which is covered by intact mucus membrane.
- It may cause serious hemorrhage the adjacent teeth may be lost.

X-ray → It shows bone destruction just below the tumor

► **Treatment:** → Wide excision including a wedge of the gum & mandible.

3- Granulomatous epulis

- It is a mass of granulation tissue around a carious tooth.
- may be covered with pus

► **Treatment:** → Excision of tissue (histological examination is essential).

4- Carcinomatous epulis

- It is an epithelioma of the gum mucous membrane.
- It may take the form of fungating mass or malignant ulcer.

► **Treatment:** → Commando's operation

5- Sarcomatous epulis

- It is fibrosarcoma.

► **Clinical Picture:**

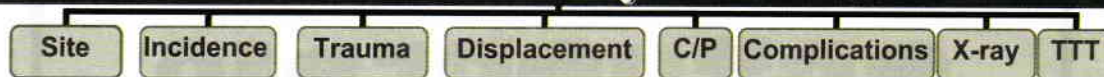
- It presents as rapidly growing painful soft mass.
- The tumor may invade the surrounding structures.
- Young age.
- Mainly blood spread.
- Bad prognosis.

9

ORTHOPEDIC SURGERY



Scheme for Any Fracture



Site

Incidence

Trauma

Displacement

- Position of distal fragment in relation to proximal one.

Clinical Picture

Symptoms

- History of trauma.
- Pain.
- Swelling.
- Limitation of movement of the affected limb (disturbance of function).

Signs

⇒ General:

- Shock.
- Hemorrhage.
- Associated injury (brain, spinal cord, viscera).

⇒ Local:

▪ Inspection:

- Swelling
- Deformity
- Ecchymosis bruises.

▪ Palpation:

- Tenderness.
- Crepitus & abnormal movement (better to be avoided).

- **Movements:** both active & passive movements are lost or limited by pain.

- **Neurovascular evaluation.**

Sure signs of fracture:

1. Deformity.
2. Length discrepancy.
3. Crepitus.
4. Abnormal movement.

Complications

- General.
- Local.

X-ray

- 2 views (A-P view, Lateral view).
- 2 occasions (before & after).
- 2 joints (proximal & distal).

Treatment

1. **First Aid :** A B C D
2. **Definitive treatment :**
 - Reduction.
 - Immobilization.

- Rehabilitation.
- Treatment of complications

Complications of Fractures

A. General Complications

- 1- **Shock:** (hemorrhagic, neurogenic & septic).
- 2- **Fat embolism:** (young patient with history of fracture long bone then on 2nd day → dyspnea, drowsiness & petechial hemorrhage).
- 3- **Complications of prolonged recumbancy:**
 - a. DVT & pulmonary embolism.
 - b. Bed sores, osteoporosis & constipation.
- 4- **Crush syndrome** → **acute renal failure.**
- 5- **Infections** → e.g. tetanus and gas gangrene.

B. Local Complications

1. Skin

A- Injury.

B- Infection.

C- Sores.

2. Muscles & Tendons

A- Injury.

B- Myositis ossificans:

- **Definition:** deposition of calcium in soft tissue i.e. heterotopic bone formation.
- It is frequent after injuries of elbow or head causing restriction of joint movement.
- **C/P:**
 - **Active stage**
 - Still pain and swelling with local heat
 - Restricted range of mobility.
 - X-ray → Cloudy shadow of ossification around joint.
 - **Quiescent stage**
 - Pain ↓ gradually.
 - Still joint stiffness
 - X-ray: circumscribed denser shadow of ossification.
- **Treatment:**
 - Prevention: early reduction of fracture and avoid massage and passive movement of the joint.
 - Curative:
 - a- During active stage → Immobilization of this joint.
 - b- After 9 months → if residual mass → resection after 9 months.

3. Blood Vessels

A- Acute Ischemia (Arterial Injury).

- **Types:** spasm, contusion, partial tear, complete tear.
- **Clinical picture:** 6Ps (Pain, Pallor, Progressive coldness, Pulseless, Paralysis & Paresthesia).
- **Treatment:** reduction of the fracture:
 - If pulse returns → follow up.
 - If no pulse (within 20 minutes) → Explore artery & repair it according to the type of injury.

B- Compartmental Syndrome:

- **Definition:** ↑ pressure (30mmHg) within a space → impairment of the circulation.

- **Incidence:** the most common site for this problem is the forearm.
- **Clinical Picture:**
 1. Tense calf or forearm muscles.
 2. Severe painful passive extension.
- **Treatment:**
 1. Prevention → fasciotomy.
 2. Curative → decompression.

C- Crush Syndrome (CS):

- **Diagnostic criteria:**
 - Crushing injury to a large mass of skeletal muscle.
 - sensory and motor disturbances of compressed limbs then become tense and swollen.
 - Myoglobinuria and/or hematuria.
- **Treatment:**
 1. ABCD, IV fluids.
 2. Forced mannitol-alkaline diuresis up to 8 L/d.

D- Volkmann's Ischemic Contracture:

- **Clinical Picture:**
 - **Early** → inability to fully extend the fingers.
→ pain in the forearm with passive extension.
 - **Late** → complete claw hand.
- **Treatment:**
 - **Early** → remove the cast if it is the cause.
 - **Late (fibrosis)** → tendon transfer.

4. Nerve Injury

- **Types:** neuropraxia, axontemesis or neurontemesis.
- **Treatment:**
 - a- **Closed** → expectant Treatment For 6 weeks (massage + electric Stimulation)
→ If failed → explore & repair the nerve.
 - b- **Open** → mark the nerve at 1st then delayed suturing (no 1^{ry} repair).

5. Bone

A-Non-union:

- **Definition:** the reaction of healing does not occur for 6 months.
- **Cause:**
 - **Presence of a gap** (the most important cause) about 2 inches or more.
 - **Loss of blood supply:** e.g. damage to nutrient vessels.
- **Clinical picture:** → Pain, swelling & abnormal range of mobility.
- **X-ray:**
 - Presence of gap.
 - Sclerosis of bone ends.
- **Treatment:**
 - Atrophied: bone graft +rigid fixation.
 - Hypertrophied: rigid fixation.

B- Delayed Union:

- **Cause:**
 - **Local factors:**
 1. Infection.
 2. Foreign body.
 3. Inadequate blood supply.
 4. Interposition of soft tissue.

- **General factors:**

1. Old age.
2. Nutrition.
3. Systemic illness as D.M.
4. Drugs as corticosteroids.

- **Orthopedic intervention factors:**

1. Improper reduction.
2. Improper /imperfect immobilization.

▪ **Clinical Picture:** persistent pain, swelling & prolonged duration of healing.

▪ **X-ray:** visible fracture line. - NO sclerosis.

▪ **Treatment:**

- Treat the cause.
- + Adequate fixation.
- ± Bone grafting.

C-Malunion:

▪ **Definition:**

- The fracture is united but with deformity due to:
 - Bad fracture.
 - Bad fixation.

▪ **Types:**

- Angular.
- Rotational.
- With shortening.

▪ **Effect of malunion:**

- Deformity is cosmetically bad.
- Interfere with the function of the limb e.g. limping.
- Predispose to osteoarthritis.

▪ **Treatment:** corrective osteotomy & plating.

D-Ischemic Necrosis:

▪ **Vulnerable sites:**

- 1- **Femoral head:** after hip dislocation or fracture neck femur.
- 2- **Carpal scaphoid** (its proximal segment).

▪ **On X-Ray:**

- Early cases → appears normal But.
- After 3 months → the avascular fragment becomes denser.

▪ **Treatment:**

- In avascular necrosis of the head of the femur use. → Austin Moore head (HEMIARTHROPLASTY).
- Of scaphoid ischemic necrosis → "vascularized bone graft from fibula".

6. Joints

A- Sudeck's Atrophy:

▪ **Etiology:** unknown But may be due to:

- Disuse atrophy.
- Sympathetic over activity.

▪ **Clinical Picture:**

- Neurotic female.
- Pain, stiffness, swelling & vasomotor changes.

▪ **X-ray:**

- Patchy osteoporosis.
- Glassy appearance of the bone.

▪ **Treatment:**

- Physiotherapy.
- Hot wax.
- Sympathectomy.

B- Traumatic Ossification (Myositis Ossificans): see before.

Treatment of Fractures

First Aid

- A. Airway → patent.
- B. Breathing → maintained.
- C. Circulation → stop bleeding by wound dressing (No tourniquet).
- D. Don't Move limb → Immobilization.
Drugs (e.g morphia for pain).

Definitive Treatment at Hospital

Resuscitation and primary survey

1. Reduction

⇒ Types:

- Closed:
 - Done under sedation/anesthesia.
 - Traction & counter-traction.
- Open: done when:
 - Closed reduction is not possible or has failed.
 - Intra-articular fractures. - Displaced epiphyseal injury.
 - Non-union. - Malunion.

2. Fixation

⇒ External:

- Types:
 - Plaster of Paris (after it: check the pulse – do x-ray).
 - Traction → skin or skeletal.
 - External skeletal fixators → In infected fractures (where internal fixation is contraindicated).
- Indications:
 - Fractures associated with severe soft tissue damage.
 - Nerve or vessel damage. - Severe comminuted & unstable fractures.
 - Fractures of pelvis. - Infected fractures.
- Complications:
 - Pin-tract infection. - Delayed union.

⇒ Internal fixation:

- Indications (P's):
 - Fractures that can not **be** reduced.
 - Unstable fractures **Prone** to displacement.
 - Fractures that unite **poorly**. - **Pathological** fractures.
 - **Polytrauma**. - Patients who **present** nursing difficulties.
- Types:
 - Screws. - Plates & screws. - Intramedullary nail.
 - Interlocking nails & screws. - Dynamic compression screw & plate.
- Choice: dependent on:
 - Patient. - Type of fracture. - Surgeon's competence.
 - Facilities available.
- Complications:
 - Infection. - Non-union. - Implant failure.
 - Re-fracture. - Malunion.

3. Rehabilitation

Physiotherapy & active movement

▪ **Methods:**

- Active exercise.
- Assisted movements.
- Functional activity.

4. Treatment of Complications

Treatment Open Fractures (Compound)

First aid

- a. Airway → patent.
- b. Breathing → maintained.
- c. Circulation → stop bleeding by wound dressing (no tourniquet).
- d. D → Don't Move limb → Immobilization.
→ Drugs (e.g morphia for pain).

Definitive Treatment at Hospital

- a. **Resuscitation** and primary survey.
- b. **GA.**
- c. **Irrigation** of wound with saline.
- d. **Wound debridement:** removal of all fragment & devitalized tissue.
 - Skin: excise 1-2mm from the edges.
 - Fascia: open tense fascia.
 - Muscles: excise dead muscles.
 - Bone: decontamination of bone by:
 - a- Curettage.
 - b- Pressure irrigation system (better).
 - Nerve → mark it for delayed repair.
 - Blood vessels → ligate, repair or graft (according to state of injured vessel).
 - Skin:
 - a- Clean (within 8 hrs) → closure without tension.
 - b- Infected (after 8 hrs) → leave it opened + sterile dressing then delayed dressing or grafting.
- e. **Immobilization:**
 - External skeletal fixation.
 - Winnet Orr technique (not done now).
 - Traction.
- f. **After care:**
 - Observe distal circulation.
 - Antibiotic.
 - Antitetanic.
 - Anti gas gangrene.
 - Later:
 - Skin: 2ry suture or graft (if loss).
 - Non union → bone graft.
 - Nerves & tendons → delayed repair.

Upper Limb

Fracture Clavicle

Commonest Site

1. At junction of medial 2/3 & lateral 1/3, as it is weakened by:
 - Change of curve & change of shape
 - Nutrient artery foramen & groove for subclavius m.
2. Fracture of outer 1/3 (rare).
3. Fracture of inner 1/3 (very rare).

Incidence

- It's the most common fracture of upper limb.
- The most common fracture in **children < 10 years**.

Trauma

- **Indirect:** fall on outstretched hand (**the commonest**).
- **Direct:** blow to the shoulder.

Displacement

- **Proximal fragment:** pulled up by sternomastoid.
- **Distal fragment:** pulled down by gravity.

Clinical Picture

1- Symptoms:

- History of trauma.
- Pain & Swelling at site of fracture.
- Limited movement (inability to move the UL).

2- Signs:

1. Inspection:

- Shoulder drop.
- Step ladder deformity = over riding.
- Position of lactating mother: The patient usually supports the elbow with the other hand & bends his head towards fracture site (to relax sternomastoid).

2. Palpation:

- Broken displaced clavicle. - Tenderness at site of fracture.

3. Movement: Limited active and passive movement.

4. Neurovascular evaluation:

- Subclavian artery & brachial plexus (C8 & T1)

Complications

- **Malunion** (most common but not of functional significance)
- Non union (uncommon due to rich blood supply)
- Shoulder stiffness
- Injury of:
 - Subclavian vessels (rare)

- Brachial plexus
 - Deformity → claw hand
 - Sensory loss → - Medial side of forearm - Medial 3 ½ fingers
 - Motor → Wasting of small muscles of the hand
- Pleura & lung

X-ray

- Antero-posterior.
- Shows: fracture & Displacement (overlap of fragments).

Treatment

- **Closed reduction:** (under GA): Usually unnecessary.
- **Immobilization:** for 3-4 weeks:
 - Arm sling (in adults)
 - Figure 8 bandage (in child)
 - + follow up radial pulse

Shoulder Dislocation

Incidence

- Is a **common** injury in **adolescents & young adult**: (in children, instead of it, fracture neck humerus occurs).
- It is common because:
 - Shallow glenoid cavity & large head of humerus.
 - Wide range of shoulder movements.

Types

- It may be anterior, posterior, or inferior dislocation (Luxatio erecta).

I. Anterior Dislocation

(The commonest 97-98%)

Mechanism (trauma)

- Fall on outstretched hand with limb in external rotation.
- Fall of heavy object on shoulder while in abduction.
- The head of the humerus lies **in front of the glenoid cavity**.
- It may be:
 - Subcoracoid (the commonest).
 - Subclavicular.

Clinical Picture

Symptoms

- History of trauma.
- Pain.
- Swelling.
- Inability to move the affected limb.

Signs

1. Inspection:

- Swelling.
- Deformity:
 - The patient holds the affected limb at the elbow by the other hand with slight abduction.
 - Loss of shoulder contour (flattening).

2. Palpation:

- Head is palpable anteriorly.
- Empty space under the acromion(empty glenoid cavity).

3. Movement: complete limitation of shoulder movements.**4. Neurovascular evaluation:** - Axillary nerve. - Axillary artery.**5. Associated injuries.****Complications****A- Early**

- **Axillary nerve injury:** (mostly neuroapraxia).
- Deltoid wasting & sensory loss over its lateral aspect.
- **Axillary artery injury** → 6Ps.
- **Associated Fractures** e.g **greater tuberosity** (usually results from excessive forces during shoulder reduction).
- **Rotator cuff tear:**
 - Includes: supra, infra spinatus, subscapularis & teres minor).
 - It complicates ant dislocation especially in elderly.

B- Late: Recurrent Dislocation (The commonest)

- **Cause:** improper immobilization → lax capsule → detached labrum.

X-ray**1. Antero-posterior view:**

- Humeral head is outside glenoid cavity - Associated fractures.

2. Lateral view: confirms that it is anterior dislocation.**Treatment****Reduction under GA**▪ **Kocher's method:**

- Outward traction.
- External rotation for few minutes.
- Internal rotation and adduction.

▪ **Hippocratic method:** (Used in neglected cases.

- Foot in axilla & do gentle traction.
- When only one person is available.

Reduction is confirmed by full range of movement under anesthesia.

Immobilization

- In adduction & internal rotation for 3 weeks in a broad arm sling or adhesive strap.

Rehabilitation

- Advise the patient to avoid vigorous movement.

Treatment of Complications**1. Axillary artery:**

- ⇒ Reduction of the fracture:
 - If pulse returns→ follow up.
 - If no pulse (within 20 minutes)→ explore artery & repair it according to the type of injury.

2. Axillary nerve:

- ⇒ **Closed** → expectant treatment for 6 weeks + massage + electric stimulation
If failed → explore & repair the nerve.
- ⇒ **Open** → mark the nerve at 1st then delayed suturing (no 1ry repair).

3. Fracture greater tuberosity:

- ⇒ **First:** reduction by putting the arm in abduction.
- ⇒ **Failed:** reduction & int. fixation.

4. Rotator cuff tear:

- ⇒ Need repair in young patient.

5. Recurrent anterior dislocation:

- ⇒ **Prophylactic:**
 - By proper treatment of shoulder dislocation.
- ⇒ **Curative:**
 - Treatment if reports > 3 times → surgical.
 - Open surgery (Bankart operation, Putti-Platt operation.)

2. Posterior Dislocation

*(Less common)***Mechanism of Injury**

- 1) Forcible internal rotation of abducted arm.
- 2) Direct blow on front of the shoulder.
- 3) Epileptic fit.
 - The head of the humerus lies behind the glenoid cavity.

It may be subacromial or subspinosus**Treatment**

- Shoulder spike.

3. Inferior Dislocation (rare)

Fracture Shaft Humerus

Displacement

	Above deltoid insertion	Below Deltoid Insetion
Upper Fragment	Adducted (subscapularis)	Abducted (deltoid)
Lower Fragment	Abducted (deltoid)	

Clinical Picture**Symptoms:**

- Traumapain.....swelling.....limited movement

Signs:

- Inspection: Patient supports the arm by the other hand
- Palpation: tenderness
- Movement: abnormal mobility at fracture site.
- Neurovascular: radial n. palsy (mostly neuropraxia).

Treatment

- Closed reduction (under GA) & U-shaped plaster.
- Open reduction and internal fixation (ORIF) → if unstable or radial nerve palsy
- TTT of Complications → as radial nerve palsy

Supra Condylar Fracture of Humerus

Site

- Metaphyseal area just above the 2 condyles of humerus.

Incidence

- Commonest** elbow injury in **children**.

Trauma & Displacement

Extension type	Flexion type
85% (the commonest✓).	15%
Fall on out stretched hands.	Fall on tip of flexed elbow.
Distal fragment is displaced posterior.	Distal fragment is displaced anterior.

Clinical Picture

Symptoms

- History of trauma.
- Pain (severe) around elbow.
- Swelling around elbow.
- Partial Limitation of movement around elbow joint (displace of fracture).

Signs

1. Inspection:

- Swelling (mass).
- Ecchymosis.
- Deformity of elbow → extension or flexion.

2. Palpation

- Tenderness around elbow.
- Crepitus (with movement) (better avoided).
- Relation between medial & lateral epicondyles & tip of elbow are not disturbed forming an equidistant Δ (DD of elbow dislocation).
- Supracondylar ridge → interrupted.

3. Movement

- Both active & passive → limited by pain.
- $\pm$ Crepitus.

4. Vascular evaluation:

- Brachial a.** may be affected causing acute ischemia (**6Ps**).
- Pain, paralysis, parathesia, pallor, pulseless & progressive coldness.**

5. Manifestations of nerve injury:

	Deformity	Sensory loss	Tests for motor
Median n.	Ape hand.	- Lat 2/3 of palm. - Palmar aspect of lat 3 ½ finger with dorsal of their terminal phalanges.	- Counting finger. - Pencil test.
Ulnar n.	Partial claw hand.	- Med 1/3 of palm + palmar & dorsal aspect of med 1 ½ fingers.	- Card test. - Froment's test.
Radial n.	Wrist & finger drop.	- Lat 2/3 of dorsum (actually snuff box only is affected).	- Ask patient to extend thumb!!

DD

	Extension Type	Post. Dislocation of Elbow
Age.	Pediatric fracture.	Usually in adults.
3 bony points at elbow.	Normal relationship.	Disturbed relationship.
Supracondylar ridge.	Interrupted.	Normal.
X-ray:	Fracture Line.	Dislocation.

Complications**The most important complications include:**

1-Acute ischemia → 6Ps.

- If not treated (within 6-8 hours) will lead to chronic ischemia.

2-Volkmann's ischemic contracture: see be4

3-Myositis ossificans: muscle hematoma with overlying ossification.

4-Malunion: in the form of cubitus **varus**.

5-N. injuries (median, ulnar or radial): mostly neurapraxia.

6-Other complications:

1. Skin → tear, blisters & necrosis.

2. Muscles & tendons → tear in brachialis m. or tendons.

3. Elbow stiffness & arthritis (on long standing cases).

X-ray

- AP & lateral Views.

- Fracture line & the displacement.

Treatment**A. First Aid: ABCD.****B. Definitive treatment at hospital:****1- Resuscitation and primary surgery.****2- Neurovasuclar examination.****3- Redcution:**

a. **Undisplaced:** no need.

b. **Displaced:**

⇒ **Closed reduction.** Under GA.

- Radial pulse observation → if absent → gradual elbow extension till pulse return.

- If extension type → pull down then anterior.

- If flexion type → pull down then posterior.

⇒ **Open reduction** (indicated in)

- Failed closed. - Open fracture.

- Vascular injury not improved after closed reduction.

4- Fixation for 3-4 wks

- Above elbow back splint. - If flexion type → in extension. - If extension type → in flexion.

- ORIF (by 2 crossing K wires) if unstable.

- External skeletal fixation (if open) (Dunlop traction).

However some surgeons prefer to fix these fractures in extension what ever the type for better correction of carrying angle and to decrease arterial impairment.

5- Rehabilitation

- Active elbow movement.
- Physiotherapy.

6- Treatment of complications:**1. Brachial a. injury:**

a- Acute ischemia (should be managed within 6-8 hour):

⇒ Reduction of the fracture:

- If pulse returns → follow up
- If no pulse (within 20 minutes) → explore artery & repair it according to the type of injury.

b- Volkmann's → tendon transfer.

2. Myositis ossificans: (avoid massage)

- Prolonged immobilization.
- If residual lesion → excision after 9 months.

3. Malunion → corrective osteotomy.**4. Nerve injury:**

a- Closed → expectant treatment for 6 weeks + massage + electric stimulation (neurapraxia or axontemesis)

→ If failed → explore & repair the nerve.

b- Open → mark the nerve at 1st by black silk.

→ Then delayed suturing (no 1st repair).

5. Open fracture → **AB + external skeletal fixation.**

Complications of Injuries around the Elbow

General

- Neurogenic shock.

Local**A- Early complications:****1. Vascular injury** → brachial artery (acute ischemia):▪ Caused by:

- Supracondylar fracture humerus (sharp lower end of upper fragment).
- Elbow dislocation.

▪ Clinical picture:

1. History of trauma.
2. 6Ps: (**P**ain, **P**allor, **P**rogressive coldness, **P**ulseless, **P**aralysis & **P**araesthesia).
3. Complications: (wet gangrene – compartmental syndrome - Volkmann Ischemic contracture).

▪ Treatment:

⇒ Reduction of the fracture:

- If pulse returns → follow up.
- If no pulse (within 20 minutes) → explore artery & repair it according to the type of injury.

2. Nerve injury:▪ Caused by:

- Supracondylar fracture humerus.
- Elbow dislocation.

▪ Affected nerve: median (commonest), ulnar, radial.

- Leads to: sensory & motor manifestations according to the injured nerve.
 - Closed → expectant Treatment for 6 months + massage + electric stimulation (neurapraxia or axontemesis)
 - If failed → explore & repair the nerve.
 - Open → mark the nerve at 1st by black silk
 - Then delayed suturing (no 1ry repair).

B- Late complications:

1. Fracture itself:

- Malunion: deformity (cubitus varus or cubitus valgus).
- Non union: in
 - Olecranon fracture.
 - Radial head fracture.
 - Condylar fracture.
 - Epicondylar fracture.

2. Vascular:

- Volkman's ischemic contracture:
 - Clinical picture:
 - Early → inability to fully extend the fingers, pain in the forearm with passive extension.
 - Late → fibrosis of flexors → complete claw hand.
 - Treatment:
 - Prophylactic:
 1. Rapid reduction of the fracture.
 2. Assessment of vascular condition of limb.
 3. Early treatment of acute ischemia.
 4. Avoid tight cast.
 - Curative:
 - ⇒ Early cases (within 8 hrs):
 - Remove the cast if it is the cause.
 - Reduce the fracture if it is the cause.
 - Vasodilators.
 - If failed explore the artery & repair.
 - ⇒ Late cases (fibrosis):
 - Mild → physiotherapy.
 - Severe → excision of dead muscles & tendon transfer.

3. Nervous: delayed ulnar nerve palsy: If malunion → cubitus valgus.

- ### 4. Muscles: Myositis ossificans: (post traumatic ossification):
- Calcified hematoma in muscle (brachialis 90%, triceps 10%).
 - X-ray: hyperdense shadow.
 - Treatment: excision after 9 months.

5. Joints:

- Elbow stiffness:
 - Due to:
 - Prolonged immobilization.
 - Intra-articular extension of fracture.
 - Surgery complicated by infection.
 - Myositis ossificans.
- Neglected dislocation.
- Recurrent dislocation & chronic inability.
- Osteoarthritis: due to improper reduction of the articular surface.

6. Skin: cast sores.

Elbow Dislocation

Incidence

- **Adults** > children.

Types

- It may be posterior (more common) or anterior.

Trauma & displacement

1. Posterior elbow dislocation:

- Fall on outstretched hand with elbow in mild flexion
- Ulna migrates backward + **fracture of coronoid**.

2. Anterior elbow dislocation:

- Direct trauma to elbow.
- Ulna migrates forward + **fracture of olecranon**.

Clinical Picture

Symptoms

- History of trauma.
- Pain.
- Swelling.
- Inability to move the elbow.
- Neurovascular injury ...etc.

Signs

1. Inspection:

- Swelling.
- Local bruises.
- The patient supports the forearm with the elbow in light flexion.

2. Palpation:

- Tenderness.
- Prominent tip of olecranon posteriorly.
- Disturbed Δ .

3. Movement: complete loss of active and passive elbow movements.

DD

- Supracondylar fractures (see table above).

Complications

- Neuro-vascular injury.

X-ray

- Dislocation.
- Exclude fracture.

Treatment

Posterior Dislocation

1. Reduction:

- Under GA + muscle relaxant → downward then forward traction.
- Check movements after Treatment.

2. Fixation:

- Above elbow cast → 3wks.
- Active exercise → 3wks.

Anterior Dislocation

- ORIF using tension band.

Treatment of Complications

- **Brachial artery:** reduction of the fracture:

- If pulse returns → follow-up.
- If no pulse (within 20 minutes) → explore artery & repair.

Colle's Fracture

Definition

- It is an **extra-articular** fracture which occurs about 1 inch above the distal end of radius with postereolateral shift & tilt of the lower segment.

Site

- Extra-articular.
- Lower radius (1 1/2 inch above articular surface of radius= 1 inch above the distal end of radius).

Incidence

- Most common in ♀ > 50 years (postmenopausal osteoporosis).

Trauma

- Fall on outstretched hand.

Displacement

- Backward and lateral shift and tilt.
- Upward impaction.

Clinical Picture

Symptoms

- History of trauma.
- Pain above wrist.
- Swelling above wrist.
- Partial** affection of movement around wrist joint.

Signs

- Inspection:** dinner fork deformity.
- Palpation:**
 - Tenderness around wrist.
 - Crepitus (better to be avoided).
 - Radial styloid process is no longer lower than that of ulna.
- Movement:**
 - Loss of active movement + painful passive movement.
- Neurovascular evaluation:**
 - Radial a. may be affected so **Allen's test** is needed.
 - Median n. injury:
 - Deformity → Ape hand.
 - Sensory loss - Lat 2/3 of palm.
 - Palmar aspect of lat 3 1/2 finger with dorsal of their terminal phalanges.
 - Tests for motor - Counting finger.
 - Pencil test.

X-ray

- AP view:**
 - Fracture line (in metaphysis of radius).
 - Lateral shift or tilt.
 - Radial shortening.
- Lateral view:**
 - Dorsal displacement or tilt of the distal fragment
 - Subluxation of inferior radioulnar joint.

DD

1. **Smith** fracture (**reverse** of Colles').
2. Chauffeur fracture (fracture styloid process of radius).
3. Scaphoid fracture.

Complications

The most important complications include:

1-Sudek's atrophy: (unknown etiology).

- Severe pain causing sympathetic Stimulation & VC → local osteoporosis → more pain (vicious circle).

2- Madlung deformity in children.**3-Radial artery injury.****4-Median nerve injury.****5-Carpal tunnel syndrome:**

- Late complication with mal-union due to compression of median n.
- As usual C/P of median n. injury (**BUT** sparing sensation of palm).

6-Other complications:-

- Skin → tear, blisters & necrosis.
- Muscles & tendons → rupture of extensor pollicis longus tendon.
- **Wrist** stiffness & arthritis (on long standing cases).

Treatment**Reduction**

1. **Closed reduction under GA:** → **Hand grip method.**
 - Traction of the hand.
 - Counter traction of humerus.
 - Push the distal fragment anteriorly & medially.
2. **Open reduction & internal fixation:** by K-wires.
 - If failed closed, vascular or n. injury.

Fixation

For 4-6 weeks

1. If isolated Colles' → below elbow cast.
2. If unstable → precut k wire fixation with the hand
Palmar flexion → 20°. Ulnar deviation → 20°.
3. If associated with fracture ulnar process → above elbow cast.

Rehabilitation

- Physiotherapy from 1st day.
- Active movement (fingers – elbow – shoulder).

Treatment of complications

- **Sudek's atrophy:** sympathectomy.
- **Radial a injury:** reduction of the fracture:
 - If pulse returns → follow up.
 - If no pulse (within 20 minutes) → Explore artery & repair it according to the type of injury.
- **N. injury:**
 - Closed → expectant treatment for 6 wks + massage + electric stimulation → If failed → explore & repair the nerve.
 - Open → mark the nerve at 1st then delayed suturing (no 1^{ry} repair).
- **Rupture of Extensor pollicis longus tendon** → transfer of extensor indices.

Fracture of Scaphoid Bone

Site

- waist of scaphoid.

Complications

- Bone: Avascular necrosis (of proximal fragment) ... Non-union... Sudek osteoporosis
- Joint: wrist stiffness.

X-ray

- informative only after 1 – 2 wks.
- *Early: hair line fracture (invisible).....Later: decalcification (visible)*

Treatment

- Plaster cast.
- ORIF (Herbert screw).

Lower Limb

Fracture Neck Femur

Blood Supply of Femur Head & Neck

- 1- Extra capsular arterial ring:
- 2- Retinacular vs:
- 3- Artery of ligamentum teres:
- 4- Nutritional vessels: from the shaft along the medulla

Incidence

- Old age > young due to senile osteoporosis.
- ♀ > ♂ due to postmenopausal osteoporosis.
- In adolescent age → slipped upper femoral epiphysis.

Trauma (mechanism of injury)

- Elderly pt. → minor trauma:
 - (As when foot catches the edge of a carpet) → **Intracapsular fracture.**
 - Direct fall over greater trochanter).
- Young patient or old → major trauma.

Classification

According to Site

- **Intracapsular:**
 - **Sub-capital** → just below femur head.
 - **Trans-cervical** → passing through the neck of femur.
 - **Basal** → at the junction between lower part of neck & greater trochanter.
- **Extracapsular:**
 - **Inter-trochanteric** → passing between both trochanters.
 - **Per-trochanteric** → passing through lesser & greater trochanters
 - **Trochanteric** → fracture of greater or lesser trochanter.
 - **Subtrochanteric** → within 5 cm below intertrochanteric line.

Displacement

- Fracture may be impacted or unimpacted.

Clinical Picture**Impacted**

- Usually no rotation, the lower limb appears to be abducted rather than adducted with only tenderness over fracture site (missed fracture).

Unimpacted Fracture▪ **Symptoms:**

- History of trauma.
- Pain in hip region → refers to groin & medial side of knee.
- Swelling in hip region.
- Inability to walk (even Inability to get up after falling).

▪ **Signs**- **Inspection:**

- Ecchymosis over the greater trochanter (**more in extracapsular**)
- Deformity →
 - 1- External rotation.
 - 2- Adduction.
 - 3- Shortening (deformity is **more in extracapsular** type)

- **Palpation:**

- Tender greater trochanter & may be rotated posteriorly & displaced.

- **Movement (straight leg raising test):** inability to raise extended leg.- **Neurovascular:**

- Sciatic n. injury:
 - a. Drop foot.
 - b. Sensory loss → - back of thigh.
- Leg & foot (except small area on medial aspect).

Complications**General**

- Complications of prolonged recumbence:
 - DVT & pulmonary embolism.
 - Bed sores.
 - Osteoporosis.
 - Renal stones.

Mortality in first 3 month is 20%

Local

- **Avascular necrosis (most common):**

Cause: occurs in **intra-capsular type** (not in extra-capsular) due to cut of blood supply of femoral head causing its necrosis & malunion.

X-ray: head will become denser then shrinks

- **Non union :**

Cause: Cutting of blood supply, Imperfect reduction, and Imperfect fix.

- **Malunion:**

- More with extracapsular fracture. - Coxa vara < 120° - Coxa vulga > 120°

- **Neurovascular injury → sciatic n. injury.**

- **Other complications:-**

- Skin → tear, blisters & necrosis.
- Muscles & tendons → tear.
- Osteoarthritis & stiffness of **hip** joint (on long standing cases).

X-ray

- A-P & Lateral view show: - Fracture. - Osteoporosis. - Displacement.

Treatment

1- First Aid: Life-saving measures.

2- TTT of fracture:

Intracapsular Fracture

- **Impacted (abduction) + non displaced (noreduction):**
 - Internal fixation by Moore pins or canulated screws.
- **Displaced (adduction):**
 - Old age > 60 yrs/neglected/complicated → Prosthetic replacement:
 - Hemiarthroplasty (Thompson or Austin Moore)
 - Young → ORIF → DHS.

Extracapsular Fracture

- ORIF (DHS)/

Treatment of Complications

- Established avascular necrosis → Austin Moore replacement or total hip replacement if there's OA. of the hip.
- Non-union:
 - Young age with viable head → subtrochanteric displacement osteotomy
 - Old age or young with non viable head → prosthesis.
- Mal-union → corrective osteotomy.
- N. injury:
 - Closed → expectant Treatment for 6 weeks + massage + electric stimulation
If failed → explore & repair the nerve.
 - Open → mark the nerve at 1st then delayed suturing (no 1^{ry} repair).
- Open fracture → antibiotics + external fixation.

Fracture Shaft Femur**Site & displacement**

- Subtrochanteric. - Mid shaft. - Supracondylar (more in adults).

	Subtrochanteric	Mid shaft	Supracondylar
Site	- 5 cm below inter trochanteric Line.	In between.	- 9 cm above femoral condyles.
Displacement			
Proximal fragment	- Abduction by <u>glutei</u> .	Forward (<u>quadriceps</u>)	- Forward by <u>quadriceps</u> .
Distal fragment	- Adduction (<u>adductors</u>)	Backward hamstring.	- Backward by <u>gastrocnemius</u> .

Clinical Picture

15% Open injuries

Symptoms

- History of trauma. - Pain. - Swelling. - Inability to move the limb.

Signs

- **General:**
 - Shock. - Associated injuries. - subtrochanteric shortening.
- **Local:**
 - Ecchymosis. - Bruises. - Abnormal range of mobility.
 - May be open fracture.

Complication**General**

- Shock.
- Complication of prolonged recumbency.

Local

- **Blood vessel injury:**
 - Femoral artery in mid shaft fracture.
 - Popliteal artery in supracondylar fracture.
- **Nerve injury:** popliteal nerve in supracondylar fracture.
- **Fracture:** - Non union. - Malunion. - Infection.
- **Joints:** stiffness of knee.
- **Muscles:**
 - Myositis ossificans. - Volkmann's ischemic contracture. - Tear.
- **Skin:** in open fractures.

X-ray (AP & Lateral view)**Treatment****Urgent Treatment (Life saving)**

- ABCD for polytraumatized patient.

At Hospital

- Resuscitation and primary survey.
- Neurovascular examination.

Treatment of Fracture (Limb Saving)

- **Subtrochanteric:** ORIF (intermedullary nail)
- **Midshaft in adults (> 15 years):** ORIF (intermedullary nail or plate and screw)
- **Supracondylar:** ORIF (angled plate and screw)
- **Midshaft in young age:** closed reduction and immobilization by:
 - ➔ Crede's method: newborn.
 - ➔ Gallow's traction: infants (< 4yrs).
 - ➔ Thomas splint: children (5-15 yrs).

Treatment of complications

- Non union → rigid fixation & bone graft.
- Malunion → osteostomy & internal fixation.
- Infection → debridement & antibiotics.

Pott's fracture

Fracture & fracture dislocation of the ankle

Definition

- Fracture lower end of tibia & fibula involving the ankle joint.

Classification**Pott's Classification**

- Unimalleolar → only one malleolus.
- Bimalleolar → medial & lateral malleoli.
- Trimalleolar → medial, lateral & posterior malleoli.

Lauge-Hanson class (most used)

- According to both Position of foot at the time of injury.
- Direction of talar movement.

Trauma

- Forcible eversion or inversion.
- Forcible rotation.
- Axial loading (falling from a height).

Types & A/E

	Abduction fracture	Adduction fracture
Trauma	Fall on everted foot.	Fall on inverted foot
1st deg	Transverse avulsion fracture of medial malleolus.	Transverse avulsion fracture of lateral malleolus.
2nd deg	1 st + oblique sharing fracture of lateral malleolus + lateral displacement of talus.	1 st + vertical fracture of medial malleolus + medial displacement of talus.
3rd deg	2 nd + fracture posterior malleolus + posterolateral displacement of talus	2 nd + fracture posterior malleolus + posteromedial displacement of talus

	Internal rotation Fracture (rare)	External rotation Fracture
Trauma	Forcible internal rotation.	Forcible external rotation.
1st deg	Oblique sharing fracture of <u>medial</u> malleolus. (Mono malleolar)	Oblique sharing fracture of <u>lateral</u> malleolus. (Mono malleolar)
2nd deg	1 st + rupture of deltoid ligament or transverse avulsion fracture of <u>lateral</u> malleolus + <u>medial</u> displacement of talus (Bimalleolar)	1 st + rupture of deltoid ligament or transverse avulsion fracture of <u>medial</u> malleolus + <u>lateral</u> displacement of talus (Bimalleolar)
3rd deg	2 nd + fracture posterior malleolus + posterolateral displacement of talus (Tri malleolar)	2 nd + fracture posterior malleolus + <u>posterolateral</u> displacement of talus (Tri malleolar)

Vertical compression fracture		
Fall on planter flexed foot	Fall on dorsi-flexed foot	Fall on plantigrade foot
Fracture posterior malleolus of Tibia & posterior dislocation of the ankle.	Fracture anterior margin of tibial articular surface & anterior dislocation of the ankle.	Diastasis of the inferior tibio fibular joint &/or central dislocation Of talus & comminution of tibial surface.

Clinical Picture**Symptoms**

- History of trauma.
- Pain & discomfort around the ankle.
- Swelling.

Signs

- Variable deformity & tenderness.
- Neuro vascular: tibial nerve & vessels.

Complications

- Malunion & non union.
- Joint complications.
- Sudeck's osteodystrophy.
- Neuro vascular: tibial nerve & vessels.
- Tendon injury.

X-ray (AP & Lateral view)**Treatment****First Aid**

- ABCD.

Emergency Treatment

- Trial of reduction for displaced fracture.
- Dislocated ankle should be reduced before X-ray.
- Open wounds & abrasions should be cleaned.

Definitive treatment

- **Fracture of lateral malleolus with no displacement:**
 - External fixation in below knee cast for 8 wks.
- **Fracture of lateral malleolus with displacement:**
 - Reduction & internal fixation.
- **Fracture of medial malleolus** → usually internal fixation
- **comminuted** → arthrodesis

Treatment of complications

E.g. sudeck's atrophy → sympathectomy.

Bone Inflammation

Acute Hematogenous Osteomyelitis

Definition

- Acute blood-borne non specific inflammation of cancellous tissue of bone & its medullary cavity

Incidence

- Child male with low standard.

Etiology• **Organism:**Gm +ve:

- Staph aureus (**The commonest**) ✓✓
- Strept. pyogenes or pneumoniae

Gm -ve:

- Salmonella (in sickle cell anemia or after splenectomy).
- H. influenza (common < 4 years of age)

• **Route of Infection:**

- Hematogenous spread from septic focus (the most common).

• Predisposing Factors

- Repeated trauma and septic foci.

Pathology

- Most common in the metaphysis of long bone.
- Commonly upper end of tibia & lower end of femur (around knee)

Mild trauma → Metaphyseal hematoma → Infection → Intera-oseous abscess → Bad

bl. Supply → Bone necrosis → Pus spread → Sinus discharge pus + sequestrum

So the characteristic pattern of pathological changes is

- 1- Hematoma
- 2- Suppuration
- 3- Necrosis (**sequestrum**)
- 4- New bone formation (**involucrum**)

Clinical Picture

a. Type of patient:

- o Usually ♂ child (may be with history of trauma)
- o **History** of septic focus e.g. sore throat – Otitis Media.

b. Symptoms:

General → very bad general condition (fever – pallor – sweating)

Local limited movement

The parents noticed that child fears to use the limb_
(**PSEUDOPARALYSIS**) → Severe pain (of sudden onset)

c. Signs:

General → High fever – tachycardia

Local

- 1- Localized tenderness over the bone (**The earliest sign**)
- 2- Redness & edema over the skin.
- 3- Sympathetic effusion of the adjacent joint
- 4- Restricted joint movement

(Unlike septic arthritis it can be moved passively with gentleness)

Complications

General

- 1- Toxemia.
- 2- Septicemia & pyemia.
- 3- Metastatic infection.

Local

- 1- Chronic osteomyelitis
- 2- Suppurative arthritis
- 3- Disturbed bone growth (deformity – shortening)
- 4- Pathological fracture

DD

- 1- Ewing's sarcoma
- 2- Cellulitis.
- 3- Septic arthritis
- 4- Rheumatic arthritis.

Investigations

i. Laboratory:

- 1- CBC: ↑TLC & ↑ESR.
- 2- The **most certain** is to aspirate pus from metaphysic.

ii. Radiological:X-ray- 1st 2 wks → -ve

- After 3 wks → sequestrum – cloaca – involucrum

Treatment*Start immediately once suspected*General:

Hospitalization, bed rest & immobilization (until inflammation subsides)

Medical

- a. Analgesic & Antipyretics
- b. Antibiotics (Early administration of high dose) → Broad-spectrum (cloxacillin, augmentin or vancomycin)

Surgery:

- If no improvement on Abs within 48 hours (*from onset of fever*)
→ Drainage of subperiosteal abscess + drilling of the cortex

TTT of complication (Chronic osteomyelitis):

- a- Antibiotics & prolonged immobilization.
- b- Sequestrectomy.
- c- Saucerization & bone graft.

TB of Spine (Pott's disease)**Definition**

- Chronic specific infection of the spine by mycobacterium tuberculosis.

Incidence**Commonest form of skeletal TB****Etiology**

- **Organism** → *Mycobacterium tuberculosis*
- **Route** → mainly 2^{ry} blood born from lung or Mediastinal LN
- **Predisposing factors** → immunocompromised

Pathology

- **Commonest site** is dorso-lumbar region due to greater mobility.
- **Sites of focus:** → 2 types
 - a) Vertebral body (**osseous type**) → more common in children
→ Destruction of bodies & inter-vertebral discs
→ Collapse & kyphosis.
 - b) Periosteum (**peri-osteal**) → more common in adult
→ Under anterior longitudinal ligament near the spinal cord
→ Ending in paralysis
- **Microscopic**
→ **Tubercles are formed of epithelioid cells & giant cells (Langerhan's cells) surrounded by lymphocytes & fibroblasts.**

Complications

1- Cold abscess:

- a) In cervical region → retropharyngeal abscess
- b) In thoracic region → parasternal & paravertebral abscess
- c) In lumbar region → Psoas abscess
(Passes under inguinal ligament with cross fluctuation)

2- T.B sinus: → due to faulty drainage of cold abscess

3- Potts' paraplegia (10%)

- a. More with **peri-osteal type**
- b. Usually in **upper dorsal spine**
 - **Early** → Due to edema → **Reversible**
 - **Late** → Due to angular kyphosis. → **Irreversible**

4- Deformity:

More with **osseous type** → Angular kyphosis

Clinical Picture

Type of patient: more common in children

Symptoms

General:

Manifestations of TB toxemia.

Local:

- 1- Severe back pain (dull aching) more at night and increase on movement.
- 2- Limitation of movement in all directions (**the most important sign** ✓✓)
- 3- Manifestations of complications.

Signs

- Limited spinal mobility (Coin test)
- Tenderness opposite the affected vertebrae
- Kyphosis - Cold abscess - Paraplegia

Investigations

1- X-ray spine (lat. View)

- Destruction of vertebral bodies and IVD. - ± Kyphosis
- Bone destruction with no new bone formation

2- CT-Scan

➤ For pulmonary TB:

- i. Blood → leucopenia with relative leucocytosis.
- ii. CXR → pulmonary lesions.
- iii. Tuberculin test (good -ve test)
- iv. Sputum examination, Zeil Nilson stain & culture on Lowenstein Jensen media.

Treatment (mainly conservative)

General:

- 1. Good diet & rest.
- 2. Anti-tuberculous drugs for 9 months
 - For 2 months. → Rifampicin + INH + Streptomycin (or Ethambutol)
 - Rest of course → Rifampicin + INH

NB. → Early Paraplegia resolves in 60% of cases

Local:**1- Conservative** → Immobilization in the active stage

- Cervical region → plastic collar.
- Thoraco-lumber region → Plaster jacket then corset.

2- Surgical drainage

(If no signs of recovery after 3 -4 weeks)

- i. Aspiration by Z technique
- ii. Open drainage + bone graft → then close without drain

Bone Tumors

Bone Secondries (50%)

Source

- Breast - Prostate - Thyroid - Lung. - Kidney - GIT

Incidence

- 2/3 cases: → from cancer breast or prostate.

Pathology

- **Macroscopic & Microscopic:** correspond to that of the 1^{ry}.
- **Site:** where red marrow is plentiful e.g.
 - Trunk bones.
 - Root bones.
- **Route:**
 - Systemic circulation.
 - Direct connection.
 - Direct invasion.

Clinical Picture

- Local bone aches (pain)
- Swelling.
- Pathological fracture.
- +/- The clinical picture of the primary tumor.

Investigations**For the primary tumor****Enzymes**

- **Acid Phosphatase:** ↑ with cancer prostate.
- **Alkaline Phosphatase:** → ↑ with bone destruction.

Radiology

- Bone scan Tc⁹⁹: - Plain x-ray:

Treatment (palliative treatment as it is grade 4)

- **Specific Treatment**
 - Hormonal → for breast & prostatic Cancer.
 - Radioactive iodine → for thyroid Cancer.
- **Radiology:** especially in vertebral metastasis
- **Chemotherapy:** CMVA
- **Surgery:** Internal fixation for path fractures or amputation.

Benign Bone Tumors

	Osteoma	Chondroma
Origin	Osteoblast.	Chondroblast
Types	1. Compact osteoma: 2. Cancellous osteoma.	1. Ecchondroma. 2. Enchondroma.
Site	• Skull • Long bone.	1. Short bone (Hands & feet). 2. Flat bones → ribs & scapula. 3. Ends of long bone.
Microscopic picture	• Osteocytes • Bone lamellae.	1. Chondrocytes 2. Cartilagenous matrix , perichondrium.
C/P	<u>Swelling which is:</u> • slowly growing • Hard dome shaped • <u>May cause:</u> - Cosmotic disfigurement. - Pressure manifestation	<u>Swelling which may:</u> • Attain large size. • Turn malignant <i>except:</i> Enchondroma of short bone of the hand & foot in children • Cause cosmetic disfigurement.
X-ray	Radiopaque dome shape shadow	Mottled shadow
ITT	Chiseling	Chiseling

Osteochondroma

Incidence

- It is common benign tumor of the bone.

Origin

- From growing epiphyseal cartilage plate

C / P

- Swelling which is: hard, well circumscribed, near joint.

X - Ray

- Mushroom like bony tumor:

Treatment

- Chiseling if causing symptoms.

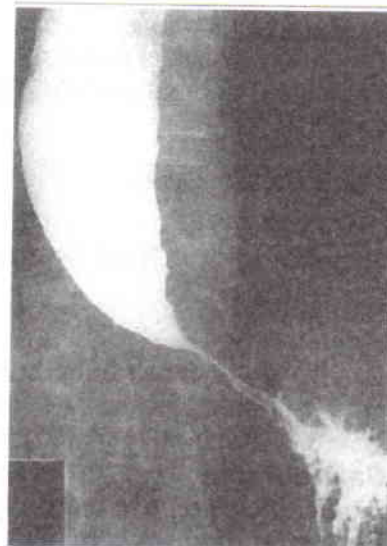
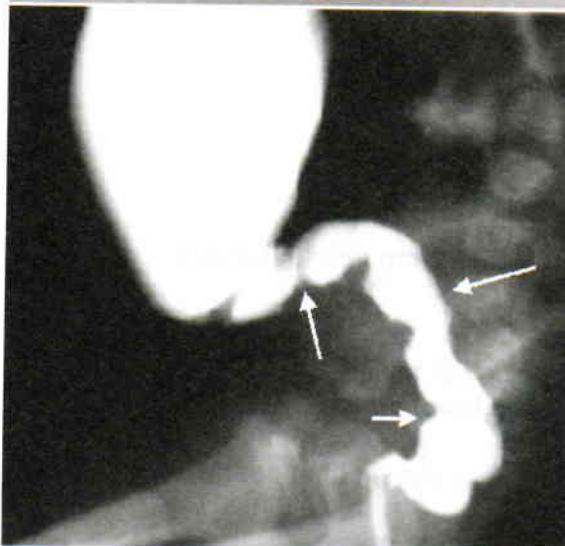
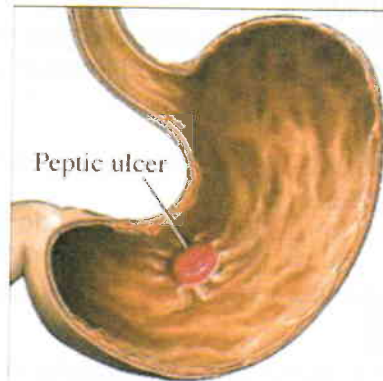
Malignant Bone Tumors

	Osteoclastoma	Osteosarcoma	Ewing's
Incidence	5-9% of all 1ry bone tumors	Most common primary malignant tumor	Mainly occur 5-15 years
Pathology			
Site	Epiphysis of long bones	Metaphysis of long bone (rule of 80)	Diaphysis & metaphysis of long bones
Cell of origin	Unkown	Osteoblasts → osteogenic	Round cell (BM reticulocytes)
Macro	- Brownish soft friable hgc areas of degeneration - Fibrous trabeculae - Thin expanded cortex	Bone destruction + formation It may be soft, fleshy, vascular (osteolytic), solid containing bone (osteosclerotic)	Soft grayish brain like tissue + areas of hgc & necrosis
Micro	- Giant cells - Stroma cells - BVs → large thin wall - Little collagen	- Malignant osteoblasts - May be → cartilage, fibrous tissue, giant cells - BVs → thin wall - hgc - necrosis	- Round cells forming rosettes around BVs → Rosette shape appearance - No matrix
Spread	- Locally malignant	Direct (course) Blood: → BLBL (80% lung metastasis) Lymphatic → Rare & Late	1. Direct 2. Blood: → BLBL (common) 3. Lymphatic → common
Clinical Picture			
Type of patient	15 - 45 yrs M > F	10-25 yrs M > F most common 1ry malignant tumor	5 - 15 yrs M > F
General	Anemia, Cachexia, Fever, Malaise, Lethargy Metastasis e.g : chest symptoms in osteosarcoma, LNs in Ewing		
Symptoms	Pain, Swelling & Pathological fracture		
	- Pain is late - Swelling - Pathological fractures common	- Pain is early - Swelling - Pathological fracture uncommon (because patient is bed ridden due to pain)	As osteosarcoma + constitutional (FAHM) D.D Acute osteomyelitis
Signs	Bony swelling which is:		

	Osteoclastoma	Osteosarcoma	Ewing's
Local Swelling	- Globular - Sharply defined edges - Egg shell crackling sensation	- Warm, tender, identified - Firm to hard with soft areas	- Warm, tender, ill defined,
Skin	- Overlaying skin → stretched	- Overlaying skin → dilated veins	- Overlaying skin → congested
LN	- No LNs	- Late affected	- Common
General	Manifestations of metastasis	- Fever, cachexia.	
Complications	1- Pathological fractures. 2- Metastasis		
Investigations For diagnosis			
1- X-ray	- Soap bubble appearance - Thin expanded cortex - Medullary plug.	<u>Ill defined destructive lesion</u> <u>New bone formation</u> - Sun ray appearance) - Codman Δ <u>Soft tissue mass around the bone</u>	Onion peel appearance
2- CT & MRI			
For staging CXR & U/S Preoperative Organ profile			
Treatment	<u>I. Important bone:</u> e.g. femur - Curettage + bone graft - Excision + reconstruction <u>II. Unimportant bone:</u> e.g. fibula → Excision with safety margin. <u>III. Inaccessible bone:</u> e.g. pelvis → Radiotherapy	<u>I. operable:</u> 1- American & French school - Above knee amputation (radical) - Chemotherapy (VAM) <u>II. Inoperable:</u> → Palliative chemo-radiotherapy, & amputation	- combination of : a. Chemotherapy b. Irradiation c. surgery - Amputation (rarely needed) - <u>Recently</u> Excision & prosthetic replacement is better than amputation

10

GIT SURGERY



ESOPHAGUS

Congenital esophageal atresia

Definition

- Failure of recanalization of foregut.

Incidence

- 1: 4,000.
- Male > female.

Etiology

- Defect in division of proximal foregut into ventral tracheobronchial tube & dorsal esophageal tube.

Types

1. Blind upper pouch & fistula of lower pouch → **COMMONEST (87%)**
2. Rare types include: atresia without fistula (7%)

Clinical Picture

- ▶ Any newborn presenting with frothy saliva should be considered as having esophageal atresia until proved otherwise.

I. symptoms:

- Continuous pouring of saliva since birth (**pathognomonic**).
- Regurgitation of food in the 1st 24 hrs. ➤ Choking, cyanosis & cough

2. signs:

- **General** → dehydration, pneumonia.
- **Local:**
 - a. Distention of the abdomen with respiration (*If fistula of lower pouch*).
 - b. Scaphoid abdomen (*If blind lower end*)
- May be presented with associated anomalies (**VACTERL syndrome**)

DD

- **Posterior choanal atresia** → Arrest at 2 cm from nostril.

Causes of Death

- Pneumonia (always occurs).
- Associated anomalies (VACTERL syndrome).

Investigations

A. **FOR DIAGNOSIS:**

1) Instrumental:

- **Catheter test:** → It will be arrested usually **10 cm** from the nostril.

2) Radiological:

- **Plain X-Ray:**
 1. ↑ fundic air bubble → fistula of lower pouch.
 2. Absent fundic air bubble → blind lower pouch.
 3. Chest: pulmonary complication.
- **Gastrographin (best):** → Shows fistula or atresia of upper pouch.
- **Fiberoptic pediatric esophagoscopy.**
- **Antenatal diagnosis by U/S:** → Dilated upper pouch

3) Laboratory: **CBC, ABG** → to assess general condition of the patient.

B. FOR ASSOCIATED ANOMALIES: e.g. Invertogram for imperforate anus, echo.

Treatment

A. Preoperative

- | | |
|-----------------------------------|-------------------------|
| 1- NPO (Nothing per os). | 3- IV fluids. |
| 2- Suction of saliva. | 4- Care of respiration. |
| 5- Prone position (↓ aspiration). | |
| 6- Avoid hypothermia. | |

B. Operative

I- Atresia with fistula :

- Rt. thoracotomy at the level of 5th Rt. rib (extrapleural approach).
- Excision of the fistula with end to end **anastomosis**.

II- Atresia without fistula:

- Gastrotomy is done at birth.
- further surgery to restore esophageal continuity is done 6 months later

+ Treatment of associated congenital anomalies

Achalasia of the Esophagus (Cardiospasm)

Definition

- Functional disorder of esophagus characterized by **failure of relaxation** of the cardia during swallowing & **absent primary peristalsis** above it

Etiology

- The exact cause is **unknown**, may be autoimmune.

Pathology

- Degeneration of **Auerbach's plexus**

Clinical Picture

Type of patient

- More often in 2nd to 4th decades & equal in both males & females.

Symptoms

1. **Dysphagia**: -Gradual onset, intermittent course & of long duration.
-Towards fluids > solids
2. **Postural Regurgitation** → alkaline, foul smelling fluid (when patient lies down during night).
3. **Halitosis** due to stasis of food → putrefaction.
4. **Retrosternal pain** (esophagitis) (stasis → infection).

Signs

- **General (in late cases only):**
- **Bad nutritional state.** - **Dehydration and Chest infection**

Complications

1. **Aspiration pneumonia.**
2. **Diverticulae** of esophageal wall.
3. **Malnutrition** → anemia & hypovitaminosis.
4. **Ulceration & hge.**
5. **Malignant changes** [□]: 3% after 20 years.

DD

(From other causes of dysphagia mainly from carcinoma)

Old ♂, rapidly progressive dysphagia of short duration & more to solids.

Investigations

1. For diagnosis:

- **Esophageal manometry:**
 - Weak peristaltic wave and Failure of relaxation of LOS.
- **Barium swallow (main diagnostic tool):**
 - Esophagus appears dilated and tortuous → **sigmoid esophagus.**
 - Lower end is pointed (narrow) → **Parrot (Hen's) - peak appearance.**
- **Plain X-Ray chest:**
 - ***Absence of gastric air bubble, chest infection, wide mediastinum.***
- **Esophagoscopy** (main aim is to exclude carcinoma: **(Biopsy + cytology in doubtful cases)**)
 - Esophagus is wide, filled with dirty water.
 - Signs of esophagitis (red esophagus).
 - Cardia is eccentric & narrow (golf hole app.)

2. For complications:

- **CBC:** anemia, hemoconcentration, leucocytosis.

Treatment

Medical Treatment

- **Isosorbide binitrate (nitrates)** or **CCBs** (usually ineffective).
- Endoscopy injection of **Botulinum toxin**: tried to relax LOS.

Forcible Dilatation by

- a) **High pneumatic pressure balloon**
- b) **Plummer's hydrostatic balloon: (obsolete)**

Surgery

- Done if all measures failed
Modified Heller's Esophagomyotomy (not extending to cardia) via abdominal or thoracic approach.

Esophageal Carcinoma

Incidence

- **Age** → 45-60 years old.
- **Sex** → Male (5% of all carcinomas) except in cervical esophagus may be more common in females.

Predisposing Factors

⇒ Chronic Irritation:

- Spicy food, smoking & spirits.
- Barrett's esophagus.
- Achalasia of esophagus.
- Plummer-Vinson syndrome.
- Postcorrosive.

⇒ Benign tumors: Papilloma or adenoma.

Pathology

Site

- Upper 1/3: 20%, Middle 1/3: 30%, Lower 1/3: 50%

Macroscopic

- infiltrating, ulcerative or cauliflower.

Microscopic

- **Squamous cell carcinoma** (40% from upper 2/3).
- **Adenocarcinoma** (60% from Lower 3 cm or On top of Barrett's esophagus).
- **Anaplastic** :(very bad prognosis).

Spread

Direct spread

- (Most DANGEROUS):to neighboring structures

Lymphatic Spread (early)

- Cervical esophagus → deep cervical LNs.
- Thoracic esophagus → posterior mediastinal, tracheal & tracheobronchial LNs
- Abdominal esophagus → left gastric & celiac LNs.

Blood Spread

- Rare & late.
- mainly to liver & lungs.

Transcoelomic

- Only in abdominal esophagus e.g. krukentburg tumour.

Clinical Picture

Symptoms

Male > 50 years old with.

- **Dysphagia**: rapidly progressive to *solids* > *fluids*, but later the patient cannot swallow his own saliva leading to continuous dripping of saliva.
- **Regurgitation** (blood stained) leads to pulmonary symptoms.
- **Excessive salivation**.
- **Loss of Appetite**.
- **Symptoms due to infiltration of adjacent structures e.g. change of voice due to infiltration of RLN.**

Signs

- **General:** Cachexia, dehydration & chest infection
- **Local:**
 - Neck: for presence of lymph nodes or mass.
 - Abdomen: for palpable hard nodular liver & ascites.

Investigations

1. **For diagnosis:**
 - Esophagoscope + biopsy + cytology
 - Barium swallow → **Rat tail appearance**, shouldering
2. **For staging:** Endoluminal sonar, CXR, CT scan, bronchoscope,
3. **For preoperative preparations:**
 - CBC, LFTs, KFTs,

Treatment

Most cases are inoperable at time of presentation.

1. Operable tumors

⇒ **Preoperative Preparation**

⇒ **Surgery:**

- 1) Tumors below the carina (tracheal bifurcation):
Ivor Lewis operation (2 phases) for middle 1/3 tumors;
- 2) Tumors above the carina:
Mc Keown operation (3 phases)
- 3) Tumors below the diaphragm (1 phase) for lower 1/3 tumours
➤ Lt **thoracoabdominal** incision, the stomach & lower esophagus are removed with Roux-en-Y esophagojejunostomy.
- 4) Nowadays many surgeons prefer to do:
Transhiatal total esophagectomy
➤ **adjuvant treatment (Chemoradiotherapy.)**
Can be effective with surgery especially with squamous cell carcinoma

2. Inoperable Tumors (60% of the patients)

⇒ **Treatment:**

- **Bypass:**
 - Gastric, colon bypass
- **Radiotherapy:** *(suitable for carcinoma in upper esophagus)*
- **Intubation:**
 - The tube is inserted by :
 - Gastrostomy (e.g. Celestin tube)
 - Esophagoscopy (e.g. Souttar tube).
- **Laser photocoagulation (YAG):**
- **Chemotherapy:** 5FU.

N.B: Barrett's esophagus:

- 10% of cases of GERD.
- Metaplasia of the lower end of the esophageal mucosa into columnar type.
- A precursor for ulcers, dysplasia, cancer in situ and adenocarcinoma.
- Regular endoscopic monitoring with multiple biopsies is essential.
- Endoscopic mucosa resection (EMR) using photodynamic therapy or argon beam coagulation may be used before progression to cancer.

Reflux Esophagitis (GERD)

Definition

- It is the reflux of acid juice (gastric contents into the lower part of the esophagus.

Causes

Sliding hiatus hernia is the most important cause

Pathology

"Belsy endoscopic grading of reflux:"

1. Grade I hyperemic mucosa.
2. Grade II intermittent superficial ulcers.
3. Grade III extensive ulceration.
4. Grade IV stricture or Barrett's esophagus.

Clinical Picture

Obese female > 40 years old

Symptoms

1. Heart burn: ↑ by lying flat and ↓ standing upright.
2. Regurgitation: especially on lying down, relieved by sitting.
3. Dysphagia due to esophageal spasm at first, but later due to stricture formation.
4. Water brush: regurgitation of acid on lying flat or bending forward.
5. Symptoms of complications (late):
Aspiration pneumonia, Barrett's esophagus,...

Signs

- **General** → Bad nutritional state.
→ Chest infection, anemia.

Investigations

A-For diagnosis:

1. Instrumental:
 - 24 hour ambulatory pH monitoring: most important one
 - Esophageal manometry

- **Upper GI endoscopy** (biopsy +cytology in doubtful cases → Belsy's grading) for columnar metaplasia "Barret's esophagus"

2. Radiological:

- **Barium swallow & meal** in 20° (Trendelenburg position):
 - Reflux of barium from stomach to esophagus.

B-For complications:

- **CBC** → microcytic hypochromic anemia due to chronic blood loss.

Treatment

I. Conservative (Main line):

- **Life style modification:**
 - Reduction of weight, small frequent meals,.....
- **Drug Therapy: (Prokinetic drugs)**
 - **Drugs to decrease gastric acidity:**
 - **Antacids:** for mild cases.
 - **H₂ blockers:** ↓ gastric acid production e.g. Ranitidine (Zantac™) 300 mg at bed time.
 - **PPI:** Omeprazole, Lanzoprazole (*reverse Barret's esophagus*)

II. Surgical:

- Approximately 10-15% of patients with GORD will be referred for consideration to have antireflux surgery.
- **Procedure:**
 - Nissen fundoplication

Sliding Hiatus Hernia

Definition

- Herniation of the cardia and adjoining part of the stomach through the esophageal hiatus in the diaphragm into posterior mediastinum.

Incidence

- Commonest of all internal hernias.

Pathology

- The gastroesophageal junction is displaced into the chest → loss of the high pressure zone → reflux of acid → esophagitis and ulceration → fibrosis → more pulling into the chest → ↑ reflux → vicious circle.

Clinical Picture

Obese female > 40 years old

Symptoms

1. Usually asymptomatic.
2. If the episodes of reflux increase the patient will have the clinical picture of GERD
3. Symptoms of complications:
 - As in GERD

4. May be part of Saint's Triad:

- Hiatus Hernia (HH)
- Diverticular disease of the colon (DD)
- Chronic calcular cholecystitis (CCC)

Signs

General: - Bad nutritional state. - Chest infection.

Investigations

1- Barium meal in the Trendelenberg's position:

- The hernia appears as a small epiphrenic bulge.
- Widening of the esophageal hiatus.

2- Perform the investigations of GORD.

Treatment

- It should be stressed that sliding hiatus hernia per se does not need treatment. Only if the symptoms of GORD are severe, then surgery will be needed.

Dysphagia

➤ See D.D book.

Postcorrosive Esophageal Stricture

Etiology

- Ingestion of caustics accidentally or attempted suicide.

Pathology

- The extent of damage depends upon the concentration of chemical & the duration of tissue contact.

Clinical Picture

History

- Ingestion of caustic material.

General

- Toxicity, high fever, neurogenic or hypovolemic shock.

Local

- Burns of the lips, mouth or tongue.
- Chest pain, dysphagia.

Complications

- Shock, chest infection, malignant transformation,.....

Treatment

Emergency Management

A B C +

⇒ At the hospital:

- **Gastric lavage is contraindicated**
- **Antishock** measures & sedation.
- **Antibiotics** (Penicillin & Flagyl).
- **No oral intake** for 1-2 weeks.
- **Steroids** (to ↓ stricture formation & laryngeal edema)
- **Plain X-Ray** abdomen & chest to detect perforation.

Definitive treatment

Repeated dilatation: if fails → surgery

– Transhiatal blunt esophagectomy + esophageal replacement by:

- Colon replacement or bypass.
- Jejunal loop.
- Stomach pull up.

STOMACH

Congenital Hypertrophic Pyloric Stenosis (CHPS)

Definition

- Thickening of the pylorus more than 8 mm. (hypertrophied)
- Normal thickness: 4 mm.

Incidence

- 0.4%, Familial (especially if the mother had CHPS), ♂: ♀ ratio is 4: 1. , common in the first infant).

Etiology

- Unknown

Pathology

- The pylorus:
 1. **Hypertrophy** in the **circular muscle** fibers.
 2. **Ends abruptly** at the duodenum and **fades gradually** towards the body of stomach.
- The stomach: First **hypertrophied** later on → **dilated** with gastritis.
- The intestine:
 - Normal collapsed and empty.

Clinical Picture

From 2-6 weeks, (**NEVER at birth**).

Symptoms

The *mother* complains that her infant has:

1. **Projectile vomiting:**
 - It is **NOT BILE STAINED** (only milk).
2. **Constipation:** dry firm stools.
3. **Failure to thrive:** progressive weight loss and dehydration.

▶ Any neonate presenting with projectile, non bile stained vomiting should be considered to have CHPS until proved otherwise

Signs

- **General:**
 - **Weight loss**, dehydration
- **Local:**

After a feed, in a good light: (Test Feed)

 - Upper abdominal distension.
 - **Visible peristalsis:** from left to right.
 - **Olive like** lump is felt in Rt. hypochondrium (**Tumor's sign**)

Complications

1. Dehydration.
2. **Metabolic alkalosis**
3. Chest infection & aspiration pneumonia.

DD Of neonatal vomiting

1. **Pylorospasm**: No palpable mass, responds well to antispasmodics.
2. **Gastroenteritis**: Fever, vomiting, diarrhea and loss of appetite.
3. **I.O**
 - **Intracranial hemorrhage**: history of trauma, scalp hematoma.
4. **Feeding problems**.

Investigations**I. For diagnosis:**

1. **U/S (most diagnostic)**: Thickening of the pyloric muscle + dilated stomach.
2. **Gastrographin study**:
 - Dilated stomach, Delayed emptying.
 - Narrow pyloric canal (**string sign**)[□].

2. For complications:

1. **Chest X-Ray**: chest infection
2. **CBC**: → anemia.
3. **KFTs**: → prerenal failure.
4. **Serum electrolytes** → ↓ Na⁺, K⁺ & Cl⁺ & paradoxical aciduria.
5. **↓ HCl with ↑ total acidity**.

Treatment**Surgical Treatment**

- ⇒ **Preoperative preparation**: *Correct general condition*
 - ⇒ Rehydration by NaCl & when urine becomes normal give KCl.
 - ⇒ TPN & Nasogastric wash of stomach by saline.
- ⇒ **Ramstedt's Pyloromyotomy**: Incision in the **anterosuperior aspect** of the pylorus (as it is the least vascular) **till the mucosa bulges**.

Peptic Ulcer

Definition

- It is ulceration of mucosa bathed in gastric juice.

Sites

1. **Duodenum**: commonest
2. **Stomach**: Less common.
3. **Rarely in**: Jejunum, esophagus, mickle's diverticulum.

Acute Peptic Ulcer

(Acute Erosive Gastritis - Acute Hemorrhagic Gastritis)

Causes

TYPES	MULTIPLE EROSIONS	TRUE STRESS ULCER
Causes	Non steroidal	• ICU patients • Sever trauma • Major burns • Endotoxic Shock
Pathology	• Occurs in the body and fundus of stomach • They are multiple , shallow and punched out • They varies in size from 1 mm to 1 cm or more • They are usually limited to the mucosa and sub mucosa	These are multiple erosions that if not recognized and treated coalesce to become the condition known as acute hemorrhagic gastritis

Clinical Picture

⇒ Symptoms:

- Hematemesis and/or melena, with epigastric pain.

⇒ Signs: Tenderness in the epigastrium.

Complications

1. Hematemesis & melena.
2. Chronicity (rare).
3. Perforation (Very rare).

Investigations

⇒ Urgent Fiberoptic Endoscopy: Visualize ulcers & the congested mucosa.

Treatment

Conservative (mainly)

A- Resuscitation

1. Ryle tube: wash by cold saline and adrenaline
2. IV line: blood transfusion, PIPs.
3. Catheter: (for follow up of urine output for fear of ARF).

B- Monitoring: Pulse, temp, BP, urine output.

Endoscopy: if localized

1. Local injection of vasoconstrictive agents.
2. Laser photocoagulation.

Surgical

A- Indication:

1. Failure of medical treatment. (very rare)
2. Complicated with perforation.

B- Methods: Generous gastrectomy.

Chronic Peptic Ulcer

Definition

- It is ulceration of mucosa & erosion of the muscle coat.
- DU:GU → 25:1 in Egypt (while in developed countries it is 4 : 1)

Etiology

- Mainly due to disturbance between gastric acidity and mucosal resistance.
- H.pylori infection is the most common cause (85% of cases in cases of DU and 75% in cases of GU).
- Risk factors include NSAIDs, smoking, irritant food and blood group O.

Clinical Picture

		DU	GU
Symptoms	Type of patient	- Male 25-40 yrs - Male : female = 5 : 1	- Male 35-45 yrs usually thin and underweight - Male : female = 2 : 1
	1. Pain		
	Type	Burning, stabbing, colic, shooting or dull aching	
	Time	a. <u>2 – 3 hrs[□] after meal</u> b. <u>Nocturnal pain</u> awakens the patient in early morning.	<u>½ - 1 hr</u> or <u>immediately[□]</u> after meals
	Site	Above the umbilicus to the Rt. of the midline.	Epigastric in the midline.
	Exciting Factors	Irritant foods, nervousness, Smoking, stress	
	Relieved by	Alkalis & Antacids	
		Food or anything buffers the gastric juice	Fasting & vomiting
	2. Vomiting	Only if pyloric stenosis occurs	Very common(as it relieves pain & may be self induced)
	3. Appetite	Good as eating relieves pain	the patient is afraid to eat (sitophobia) as it induces pain
Signs	4. Body wt.	Gain weight	Loss of weight
	5. Hematemesis & melena	+ve (<u>more melena</u>)	+ve (<u>more Hematemesis</u>)
	1. Anemia	+ ve	+ ve
	2. Pointing sign	The patient can localize site of pain exactly by one finger	
	3. Tenderness	Above the umbilicus to the Rt. of the midline	epigastric in the midline

DD1. Gastric Lesions:

- Acute ulcer
- Gastritis
- Carcinoma
- Hiatus hernia.

2. Wilkie's Triad: Chronic peptic ulcer + chronic cholecystitis + chronic appendicitis.3. Other Causes of Dyspepsia.**Investigations**A- For diagnosis:➤ Upper GI endoscopy (investigation of choice):

1. For diagnosis.
2. For follow up.
3. 4 quadrant punch **biopsy** + brush cytology. (only in GU).

➤ Barium meal:

	DU	GU
Ulcer niche	In the duodenal cap	on the lesser curvature.
Deformity	(trifoliate deformity)	Ulcer niche with opposite notch
Post evacuation	Ulcer crater → A flake of Barium remains in the ulcer niche & The mucosal folds are seen radiating toward it	
Complications:	Soup dish appearance (in pyloric stenosis)	hour glass or tea pott app.

- CXR: Air under diaphragm → perforated peptic ulcer.

B- For complications:➤ For bleeding➤ CBC:

- Microcytic hypochromic anemia due to chronic blood loss.

➤ Stool analysis:

- By benzedine test for occult blood loss.

➤ In recurrent cases, we should search for the cause:

- H. Pylori → biopsy, serology & urease test

Treatment

TTT of uncomplicated peptic ulcer is mainly medical

MedicalA- LIFE STYLE MODIFICATION:

1. Rest: Physical & mental rest (sedatives might be needed)
2. Small frequent meals: about 5 times a day
3. Avoid irritant foods → spicy etc.
4. Avoid irritant drugs → aspirin, cortisone etc.
5. Avoid → smoking, alcohol, coffee, tea and cola.

B- Drugs:**H₂ blockers:**

1. Cimetidine 800mg at bed time
2. Ranitidine 150mg twice daily.
3. Famotidine 20mg twice daily.

Proton pump inhibitor:

- Omeprazole 20mg in the morning.

- **Antacids:** e.g., Mg hydroxide.
- **Coating agents:** Sucralfate and Bismuth.
- **Drugs enhance mucosal barriers:**
 - As Carbenoxelone & Prostaglandins.
- **Treatment of H. pylori by triple therapy for 10 days:**
 - Omeprazole + Metronidazole + Clarithromycin.

Surgical**A- Indications:**

1. Failure of medical treatment. (for 6 wks in GU or 6 months in DU)
2. Complications.
3. Non-compliant patients.
4. Patient cannot pay for the drug.
5. If malignancy can't be excluded in GU.

B- Methods:**1. Duodenal ulcer:**

- Truncal vagotomy .
- Selective vagotomy .
- Highly selective vagotomy.
- Lesser curve seromyotomy.
- Vagotomy and antrectomy (associated with the lowest recurrence rate)

2. Gastric ulcers:

- Partial gastrectomy & reconstruction (by Billroth I or II –polya or polya with valve) is the standard procedure.

Complications of PU

A-Acute Complications:

⇒ That occur during periods of ulcer activity:

1. Perforation.
2. Bleeding.

B-Chronic complications:

⇒ That occur insidiously:

1. Fibrous contractures of ulcer produce pyloric stenosis or hourglass stomach.
2. Penetration.
3. Malignant transformation of GU.

1 - Perforation

Acute Perforation

Incidence

- More common in **DU (90%)**
 - More common in **males**.
 - More common in **anterior wall ulcers**.

Predisposing Factors

- All factors which causes exacerbation as stress, NSAIDs or heavy meal.

Stages

- A- Stage of Chemical Peritonitis: usually short and the patient is not seen in it
- There is a sudden rupture of the ulcer base → gastric or duodenal contents in peritoneal cavity → chemical peritonitis.
- B- Quiescent Stage (Stage of Illusion):
- It occurs 3-6 hours after the perforation.
 - Reaction of the peritoneum against chemical agent → production of large amount of alkaline fluid and bringing antibodies.
- C- Stage of Septic Peritonitis:
- It occurs 6-12 hours after the perforation.
 - Bacterial invasion → pus formation → septic peritonitis.

Clinical Picture

- ➔ **History: ulcer dyspepsia (but it may be silent till perforation occurs)**
- ➔ **Symptoms:** sudden severe upper abdominal pain that becomes generalized (stage of chemical peritonitis) then it ↓es (quiescent stage) then ↑es with FAHM (stage of septic peritonitis).
- ➔ **Signs**
 - **General:**
 - Pallor, sweating, subnormal temp, rapid weak pulse, hypotension.
 - **Local:**
 - Inspection: rigidity.
 - Palpation: guarding, tenderness& rebound tenderness.
 - Percussion: ↓liver dullness, shifting dullness.
 - Auscultation: ↓ intestinal sounds.
 - **P/R:** fullness in Douglas pouch in females or rectovesical pouch in males.

Investigations (URGENT)

- ▶ Ba meal → chemical peritonitis
- ▶ Endoscope → difficult to diagnose

1. Radiological:

- **Plain X-Ray abdomen erect** → air under the diaphragm.
- **Gastrografen meal:** ensure escape of the dye through the perforation.

2. Laboratory:

- CBC, KFTs and electrolytes.

Treatment (Urgent operation after good preparation)**1. Resuscitation & Monitoring:****a- Resuscitation:**

- **Ryle:** continuous aspiration.
- **IV** (fluids, blood, antibiotics, analgesics, H₂ blockers, omeprazole).
- **Catheter**

b- Monitoring: of BP, pulse, urine output, CVP, electrolytes and pH.**2. Emergency Operation: (the simplest procedure is done)**

⇒ *Peritoneal toilet then according to condition of the patient:*

Bad general condition or late presentation (> 12 hr):

- 1- Simple closure with **omental patch** (Graham's method) with postoperative antiulcer TTT.
- 2- in GU, biopsy must be taken.

Good general condition or early presentation (< 12 hr):**Definitive Surgery**

1. DU → Vagotomy & drainage.
2. GU → partial gastrectomy & reconstruction.

3. Postoperative Care:

- Medical treatment of the ulcer for patients who do not undergo definitive surgery.

Subacute Perforated Peptic Ulcer

- This is a small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed
- It gives the same early signs but much reduced.
- If diagnosed correctly it can be treated conservatively

Chronic Perforation of Peptic Ulcer (Penetrating Peptic Ulcer)

- Means erosion of an adjacent structure as the pancreas.
- It is suspected when the pain becomes more or less persistent and referred to the back

2-Bleeding Peptic Ulcer

Incidence

- One of the most common causes of hematemesis.

Pathology

1. **Mild** → Bleeding from the granulation tissue.
2. **Moderate** → Due to erosion of small vessels.
3. **Severe** → Due to erosion of a large vessel as **gastroduodenal artery**☐.

Clinical Picture

Symptoms

- **Past history of Ulcer dyspepsia may be present then:**
Hematemesis, melena or fresh bleeding per rectum in severe cases.

Signs

- **General:**
 1. Anemia in case of repeated minor bleeding.
 2. Hypovolemic Shock (sweating, pallor, weak rapid pulse).
- **Local:** Epigastric tenderness.

DD

From other causes of hematemesis

Complications

1. Severe shock .
2. Renal failure.
3. Respiratory complication.

Investigations

(Urgent investigations after resuscitation)

1. Upper GI endoscopy
 - For diagnosis and exclusion of other causes of hematemesis.
2. Laboratory:
 - **CBC**
 - **Electrolytes.**
 - **KFTs.**

Treatment

(Most of cases stop bleeding spontaneously due to clot formation at base of ulcer)

1. **Conservative ttt. In hospital (Resuscitation + Monitoring):**

Resuscitation:

Ryle tube: NG lavage by cold saline.

- IV line:(fluids, blood, antibiotics, analgesics, H2 blockers, omeprazole).
- Catheter

Monitoring: of BP, pulse, urine output, CVP, electrolytes and pH.

2. **Once the patient is stabilized → urgent endoscopy**

Diagnostic

Exclude other causes of hematemesis by alcohol or adrenaline in ulcer base.

Therapeutic

Laser coagulation, thermal coagulation, injection

3. Surgical Treatment:➤ Indications:

1. Failure of conservative treatment.
2. Severe bleeding from the start of about 2L or more.
3. Continuous bleeding.

➤ Methods:

- If GU → partial gastrectomy & reconstruction.
- If DU → Truncal vagotomy + pyloroplasty + under running sutures for hemostasis. (Gastroduodenal artery may be ligated)

3- Fibrous Contracture**Pyloric Stenosis****Pathology**

It is actually a duodenal stenosis

1. **Pylorus** → stenosed (spasm then fibrosis).
2. **Stomach** → hypertrophic dilatation with gastritis due to stasis.
3. **Intestine** → normal.

Clinical Picture**Symptoms**

- **Vomiting:** Projectile, **contains food of previous day, not bile stained**, foul odour from fermentation, characteristically in the evening
- **Progressive weight loss & constipation.**

Signs

(We have to exclude malignancy)

- **General:** Dehydration, chest infection, weight loss.
- **Abdominal:**
 - **Inspection:** upper abdominal fullness, visible peristalsis from left to right
 - **Auscultation:** succussion splash.

Complications

See CHPS

Investigations1. Radiological:

- **Barium meal:** soup dish appearance, **dilated stomach** with narrow pylorus & delayed gastric emptying.

2. Instrumental:

- **Upper GI endoscopy** (Main value to exclude malignancy ± biopsy): Dilated stomach with atrophic gastritis & failure of passage through pylorus.

3. Laboratory:

- **CBC** → anemia.
- **KFTs** → prerenal failure.
- **Serum electrolytes** → ↓ Na⁺, K⁺ & Cl⁻ & paradoxical aciduria.

Treatment*(Only surgical)***Preoperative**

- a) *Correction of the general condition* of the patient.
- b) *Gastric lavage through Ryle's tube.*

Operative

- a) *Truncal vagotomy & gastrojejunostomy is the standard treatment*
- b) *In the elderly or unfit patients:*
Gastrojejunostomy alone or endoscopic balloon dilatation
- c) *Partial gastrectomy.*

Hour glass stomach

- Due to cicatrized saddle shaped ulcer on the lesser Curve
- It presents by symptoms of gastric obstruction but there will be early satiety loss of weight
- Endoscopy and barium meal will diagnose it
- TTT is by Partial gastrectomy

4-Recurrent Peptic Ulcer 3%**Etiology**

Persistent Hyperacidity	Inadequate Surgical Procedures	Use of Ulcerogenic Drugs
1. Zollinger Ellison \$ 2. Hyperparathyroidism	1. Incomplete vagotomy or crimial nerve of Grassi. 2. Inadequate gastrectomy 3. Inadequate drainage	NSAIDs Cortisone

Sites

1. Gastric.
2. Stoma.
3. Jejunal.
4. Recurrence of the same ulcer.

Clinical Picture

- Recurrent Dyspepsia + other symptoms as peptic ulcer.

Investigations

- Endoscopy, barium meal, investigations for the cause e.g., serum calcium.

Treatment**A. Treatment of the cause****B. Treatment of the Ulcer**➤ **Medical:**

(As peptic ulcer).

➤ **Surgical:**

1. If medical treatment fails.
2. If after vagotomy → antrectomy
3. If after gastrectomy → higher gastrectomy + vagotomy.

Complications of Partial Gastrectomy

A- Operative Complications:

1. Complications of anesthesia.
2. Primary hemorrhage.
3. Injury of important structures.
4. Infection

B- Postoperative Complications:

I. Early Complications:

1. Hemorrhage:
2. Stomal Obstruction due to:
3. Leakage from the anastomosis:
4. Duodenal Stump Blowout:
 - Serious complication but infrequent. Responsible of 60% of mortality after gastrectomy.
 - Timing: 4th day.
 - Causes:
 - Sutures taken in inflamed or scarred duodenum.
 - Afferent loop obstruction.
5. Paralytic Ileus:
6. Acute Gastric Dilatation,
7. Burst Abdomen: (see Hernia).
8. Pulmonary Complications:
9. Subphrenic Collection:

2. Delayed Complications:

1. Postgastrectomy Syndromes:
 1. Nutritional Syndrome:
Weight loss, anemia, steatorrhea and calcium deficiency.
 2. Afferent Loop Syndrome:
 1. Cause: stomal edema, kink of afferent loop, narrowing by sutures of gastrojejunostomy, retrograde intussusception.
 2. TTT: surgical conversion of anastomosis to Roux-en-Y loop.
 3. Postcibal Syndrome (Dumping Syndrome)

Postcibal Syndrome (Dumping Syndrome)	
Early Dumping	Late Dumping
1- Hypovolemia	1- Hypoglycemia
2- Within ½ hour after meal	2 - 3 hours after meal
5- Prevention	
▪ Small frequent meals, rich in proteins and fats and less in CHO ▪ Liquids allowed only in between meals	▪ Small frequent meals , low CHO diet ▪ Oral hypoglycemic (Tolbutamide 500mg before meals)
6- Treatment:	
▪ Assuming the supine position ▪ Polya converted to Reux-en-Y to delay emptying	▪ Avoid high CHO diet ▪ Somatostatin

2. Recurrent Ulcer (3% after 2 years)

3. Gastro-jejuno-colic fistula
4. Intestinal Obstruction
5. Reflux Alkaline Gastritis (Biliary Gastritis)
6. Diarrhea

▪ **Causes:**

- Rapid gastric emptying.
- Vagal denervation of small intestine.
- Malabsorption of bile salts.

7. Gall bladder stones:

8. Increase risk of gastric carcinoma after 10 years.

Carcinoma of the Stomach

Incidence

- Males > 45 yrs.

Predisposing factors

Chronic lesion	Chronic irritation	Hereditary
2. <i>atrophic gastritis</i>	- Spicy food.	- Family history
3. <i>Biliary gastritis</i>	- Spirits.	- Female > Male
4. <i>Benign gastric tumors</i>	- Smoking.	- Fatal
5. <i>chronic GU</i>	- Smoked fish, ↑ salt intake.	- Blood group A

Pathology

I- Macroscopic			
Site			
Lower 1/3 [□] (Pylorus)	Middle 1/3	Upper 1/3	
60%	15%	25% (↑nowadays up to 60%)	
Types			
Site Gross Differentiation	1- Cauliflower	2- ulcerative	3- Infiltrating (Linitis plastica)
	Body&fundus	Pylorus or lesser curvature	Starts in antrum → extends upward → involve the stomach diffusely
	Fungating mass	Ulcer with raised everted edge&necrotic floor	marked thickening of the wall and reduction of the lumen with intact mucosa
	well	moderate	Poor
4- Colloid type:			
All layers of the stomach are infiltrated by areolar tissue containing transparent gelatinous substance. It is the commonest cause of Krukenberg tumor of ovary [□] .			
II- Microscopic			
Adenocarcinoma 95%		Squamous cell carcinoma 4%	Anaplastic 1%
Columnar cell adenocarcinoma	Colloid adenocarcinoma (Bad prognosis)	In Cardia&Fundus On top of metaplasia	Undifferentiated tumor

Spread

1. Direct: to the surroundings
2. Blood:
 - Mainly → liver via portal circulation.
 - Rarely → lungs, bones, brain via systemic circulation.
3. Lymphatic:
 - Early: to celiac & superior mesenteric LNs.
 - Late: → Virchow's LNs (Troisier's sign).
- Transcelomic: either by Seeding or Retrograde peritoneal lymphatic spread e.g:
 - *Krukenberg's tumor: metastasis to ovaries in young female.*
 - *Blumer's shelf: metastasis to Douglas pouch.*

Clinical Picture

► *Male > 40 yrs, with unexplained dyspepsia resistant for TTT for more than 2 weeks*

1. Dyspepsia group:
 - Usually, male above 40 yrs with:
 - dyspepsia, anorexia & dislike to meat
 - vague sense of discomfort after meals not responding to medical treatment > 2 weeks.
2. Cachexia group: (insidious group)
 - Loss of weight, asthenia & anemia.
3. Mass group:
 - An epigastric mass, about **30% of these patients are inoperable.**
4. Obstructive group:
 - At the cardia → dysphagia.
 - At the pylorus → vomiting.
5. Metastatic group:
 - A hard irregular liver due to secondaries, jaundice, malignant ascites
 - Enlarged Virchow's LNs (Troisier's sign).

NB: Bleeding (→ hematemesis and melena)

Is Uncommon and usually after TTT due to necrosis of the tumor

Investigations1. For diagnosis:

- Upper GI endoscopy+ biopsy
- Barium meal: (accuracy 75%)
 - If cauliflower mass: **Irregular filling defect**
 - If malignant ulcer: **Ulcer niche** (> 2 cms. Or Outside the ulcer bearing area) or **carmen's meniscus sign**
 - If infiltrating: **Linitis plastica** (leather bottle stomach)

2. For staging:

Abdominal U/S, CT scan, CXR, bone scan

3. For follow up:

- Tumor markers:as CEA or CA 19-9.

4. For preoperative preparation:

CBC, LFTs, KFTs....

Treatment**Operable Cases**A. English school:

- ⇒ Upper 1/3: *Esophagogastrectomy+esophagojejunostomy by Roux-en-Y.*
- ⇒ Middle 1/3: *Total radical gastrectomy +Esophagojejunostomy by Roux-en-Y*
- ⇒ Lower 1/3: *Lower radical partial gastrectomy +gastrojejunostomy by Polya or Polya with a valve*

B. Japanese school:

Total radical gastrectomy whatever the site of the tumor+
Esophagojejunostomy by Roux-en-Y

Inoperable Cases➤ Resectable:

Palliative partial gastrectomy.

➤ Irresectable:

1. Pyloric obstruction → *Palliative gastrojejunostomy* or *expandable metal stent.*
2. Cardiac obstruction → *Celestin tube* or *osphagojojunostomy.*

Gastrointestinal stromal tumors (GIST), formerly leiomyoma.

Pathology

1. Site: Common in the stomach and small intestine
2. Origin: from Cajal cells that lie within the stomach wall.
3. may have a pseudocapsule so recurrence is common after surgery.

C/P

- abdominal mass, hematemesis or dyspepsia.

Treatment

- Local gastric resection.

N.B. Gleevac is tumor specific monoclonal antibody against some tyrosine kinases of the tumor. It has shown excellent clinical results

GALL BLADDER

Gall bladder stones

Incidence

- 20% of females more than 40 years (female, fatty, fertile, forty or fifty & flatulent)⁷.
- Female: male → 3:1, but this female predominance is not present after 60 years of age and in pigment stones.

Etiology

A- Cholesterol and mixed gall stones

1- Disturbed bile salts cholesterol ratio.

➔ The following factors may disturb this ratio :

- i. Reduced bile salt pool
 - Malabsorption of bile salts in the terminal ileum in Crohn's disease, small bowel resection
 - Diminished hepatic synthesis in liver disease
 - Oestrogens reduce the concentration of bile salts in bile
- ii. Increase cholesterol synthesis in obesity, high dietary fat and high caloric diet

2- Stasis of bile

- Progesterone causes relaxation and impaired emptying of the gall bladder.
- Oestrogen, oral contraceptives and repeated pregnancy
- Following truncal vagotomy due to denervation of gall bladder
- Diabetes mellitus
- Obesity
- Long term parenteral nutrition.

B- Pigment stones

- 1- Haemolytic anaemias
- 2- Liver cirrhosis.
- 3- Infection : plays a role in the formation of brown pigment stones.

Types

	Cholesterol	Mixed	Black pigment	Brown pigment
Incidence	8%	80% [□]	Patient with hemolytic anemia & cirrhosis	
Constituents	Pure cholesterol	- Cholesterol (60%) CaCO ₃ , Ca bilirubinate - Bile salts, bile pigments - Phospholipids	Calcium bilirubinate	- Major → calcium bilirubinate. - Others → calcium malmitate and cholesterol;
Number	- Usually single [□] Sometimes multiple	Multiple	multiple	multiple
size		0.5-2.5cm.	<2.5 cm	<2.5 cm
Shape	Rounded or oval	Faceted	Multiple specula	laminated
Surface	Mamillated	Smooth		
Color	yellow	Yellowish	Tarry black	brown
Consistency	Hard & floats	Hard & sinks		
Cut section	No nucleus Radiating	Nucleated + laminated		
X-Ray	Radiolucent [□]	Radio-opaque [□] in 15% of cases	Radio opaque in 50 % cases	Radio opaque

Clinical Picture**Type of patient**

- 6F (Female, Forty or Fifty, Fatty, Fertile, Flatulent)

Asymptomatic (most common[□] 70%)**Symptomatic gall stones**

- **Biliary colic:** (Sudden onset of colic in the rt. hypochondrium) (about 15% of cases)
 - Radiates to: **Rt. shoulder, the back**
 - Severe attacks may be accompanied by nausea & vomiting.
 - If persists → acute cholecystitis. (Less than 5-6 hrs.)
- **Biliary dyspepsia:** (abdominal distention, excessive eructation after fatty meals)

On examination: tenderness in the right hypochondrium and Murphy's sign may be +ve.

Complications

1. Obstruction of:

- a. Hartmann's pouch or cystic duct → biliary colic, acute cholecystitis, mucocele, empyema.
- b. CBD → obstructive jaundice
- c. Ampulla of Vater → pancreatitis.

2. Migration:

- a. Through cystic duct: biliary colic & transient jaundice.
- b. Through fistula:
 - i- Cholecysto-choledochal fistula: Mirizzi Syndrome[□].
 - ii- Cholecysto-duodenal fistula:
 - Gall stone ileus[□] (due to obstruction at terminal ileum)
 - Usually in old females
 - Stop 2 feet from ileocecal valve
 - iii-Cholecysto-colic fistula:
 - Passes with stools.

3. Infection: cholecystitis, ascending cholangitis, pancreatitis.

4. Ulceration: hemobilia (very rare).

5. Metaplasia: squamous metaplasia followed by carcinoma[□] (very rare).

Investigations

A. Abdominal U/S: (inv. Of choice[□])

- Detects size & thickness of GB & presence of the stone (in 98% of cases).

B. Plain X-Ray:

- Only 10-15%[□] are radio-opaque

Treatment

A. Asymptomatic Gall Stones: No treatment (wait & watch[□]) except in:

- Diabetic patient
- Congenital hemolytic anemia
- Patients who will undergo Bariatric surgery for morbid obesity.
- Young fit patient as life span is long and there high chance for development of complications.
- Porcelain gall bladder → per cancerous

B. Symptomatic Gall Stones:

Cholecystectomy is the standard treatment[□]

Acute Calcular Cholecystitis

Microorganisms

- Usually by gram –ve e.g. E.coli, Proteus & Klebsiella.

Routes of Infection

- Direct extension → ascending from duodenum.
- Blood spread → from chronic septic focus (via cystic artery).

Pathogenesis

- Obstruction of cystic duct or neck of GB by stone → stasis → infection.

Pathology

Types

- Catarrhal, suppurative or gangrenous.

Macroscopic Picture

❖ Gall bladder:

- **Shape:** distended.
- **Lumen:** may contain stones.
- **Wall:**
 - **All layers:** edematous & hyperemic ± multiple micro-abscesses.

Clinical Picture

Characteristic 6F patient with past history of gall stones (biliary colic, biliary dyspepsia)

Symptoms

- **General:**
FAHM (fever is high).
- **Local:**
 1. **Pain:**
 - At first: It is diffuse colicky upper abdominal pain.
 - Then it becomes dull-aching, localized to right upper quadrant.
 2. Usually there is **nausea**, sometimes **vomiting**.

Examination

- **General:**
 - A characteristic patient: 6F
 - High temperature (fever) & tachycardia.
 - Jaundice rarely large stone may be impacted in Hartman's pouch and compress bile duct (MIRRIZI syndrome)
- **Local (maximum on the Rt. hypochondrium):**
 - *Signs of acute abdomen* → marked at Rt. Hypochondrium.
 - ✓ **Inspection:** rigidity.
 - ✓ **Palpation:** tenderness, guarding, rebound tenderness.
- **Special signs:**
 - ✓ **Boas' sign**[□]: An area of hyperesthesia between Rt. 9th & 11th ribs posteriorly.
 - ✓ **GB mass:** difficult to palpate due to overlying tenderness and rigidity.

Murphy's sign may be detected

Consequences

- **Resolution:** in most cases.
- **Complications:**

General

- Toxemia, septicemia, Pyemia.

Local

1. **Empyema and mucocele.**
2. **Chronic change.**
3. **Spread of infection.**
4. **Perforation (Rupture): Rare.**
5. **Jaundice (due to affection of the CBD).**
6. **Emphysematous cholecystitis.**

Investigations

A. For diagnosis

1. Radiological:

- **U/S:** (investigation of choice)

➤ **Gall bladder:**

- Stones obstructing cystic duct with sensitivity reaching 98%
- Distension. - Thickened wall.
- Serosal edema. - Micro abscesses.

➤ **CBD:**

- Diameter of the CBD (normal 0.6 mm)
- Stones.

- **HIDA:**

➤ **Scanning of the liver & gall bladder:**

- Is superior to U/S (**most accurate but not practical**).

Treatment

1. Treatment of acute cholecystitis.
2. Treatment of complications.

Treatment of Acute Cholecystitis

⇒ **According to time of presentation:**

A- **Within 1st 3 days: Cholecystectomy**

15% of patients undergoing cholecystectomy have had CBD stones.

- **Provided that:**

- 1- Sure diagnosis.
- 2- Good surgeon.
- 3- Fit patient.

B- **After 48 hours: Medical treatment**

1. **Put the patient in modified Fowler's (semi-sitting) position.**
2. **Feeding:** Stop oral feeding & IV fluids.
3. **Ryle:** tube suction.
4. **Hot application:** to the Rt. hypochondrium.
5. **Antispasmodics:** to relax the sphincter of Oddi.
6. **Sedatives.**
7. **Antibiotics:** (start once diagnosis is made)
8. **Observation:** Pulse, temp, severity of pain & early manifestations of complications

- ⇒ **If improved** → cholecystectomy after 6 weeks.
 ⇒ **If not improved** → cholecystectomy and if difficult → cholecystostomy till improvement then cholecystectomy after 6 weeks.
 ⇒ **NB: it is the trend now to treat acute cholecystitis by early surgery.**

Obstructive jaundice

Definition

- Obstruction of the bile flow to the duodenum with rise of direct bilirubin & bile salts in the blood.

Etiology

A. Intra-hepatic (non-surgical)

- Viral hepatitis (Watson \$), liver cirrhosis, sclerosing cholangitis.

B. Extrahepatic

1. Lumen:

- Calcular obstructive jaundice.
- Parasitic infestation: as ascaris & fasciola.

2. Wall: (stricture of CBD due to)

- Congenital: biliary atresia and choledocal cyst.
- Acquired:
 - Inflammation → sclerosing cholangitis
 - Trauma → post-operative.
 - Malignant stricture as cholangiocarcinoma.

3. Pressure from outside

- 1ry tumors: cancer head of pancreas and periampullary carcinoma.
- Secondaries in LN: in porta hepatic.

Clinical picture

- Jaundice.
- Pale clay stool, steatorrhea, and dark frothy urine.
- Bleeding tendency, bradycardia and pruritis.

	Calcular OJ	Malignant OJ
1. Type of patient	6 F	Elderly male
2. Jaundice	Slowly progressive, not severe intermittent,	Gradual, rapidly progressive or intermittent
3. Stool	Clay colored	Clay colored or silvery stool
4. Pain	- Recurrent attacks of dull aching pain - Rt. Hypochondrium → Rt. Shoulder	- May be absent - Boring - Epigastrium → Back
5. Weight loss	-ve	Marked
6. LNs	-ve	+ve (Virchow's LNs)
7. Liver	Enlarged, not tender	Enlarged, not tender or enlarged, nodular & tender
8. U/S	Usually fibrosed gall bladder with stones	- distended gall bladder with thin wall
9. fever	- may be present	- usually absent

Investigations

A. Laboratory

- LFTs:
 - Bilirubin: ↑ serum bilirubin mainly direct fraction[□]. It usually does not exceed 10mg/dl and its level may fluctuate.
 - Alkaline phosphatase, γ GT and 5-nucleotidase are elevated
 - PT: Prolonged, improved by IV vitamin K
- Stool:
 - Clay colored, bulky offensive.
- Urine:
 - Bilirubin: ↑ direct > 10 mg%[□].
- BUN & creatinine: ↑ in hepatorenal failure.

B. Radiological

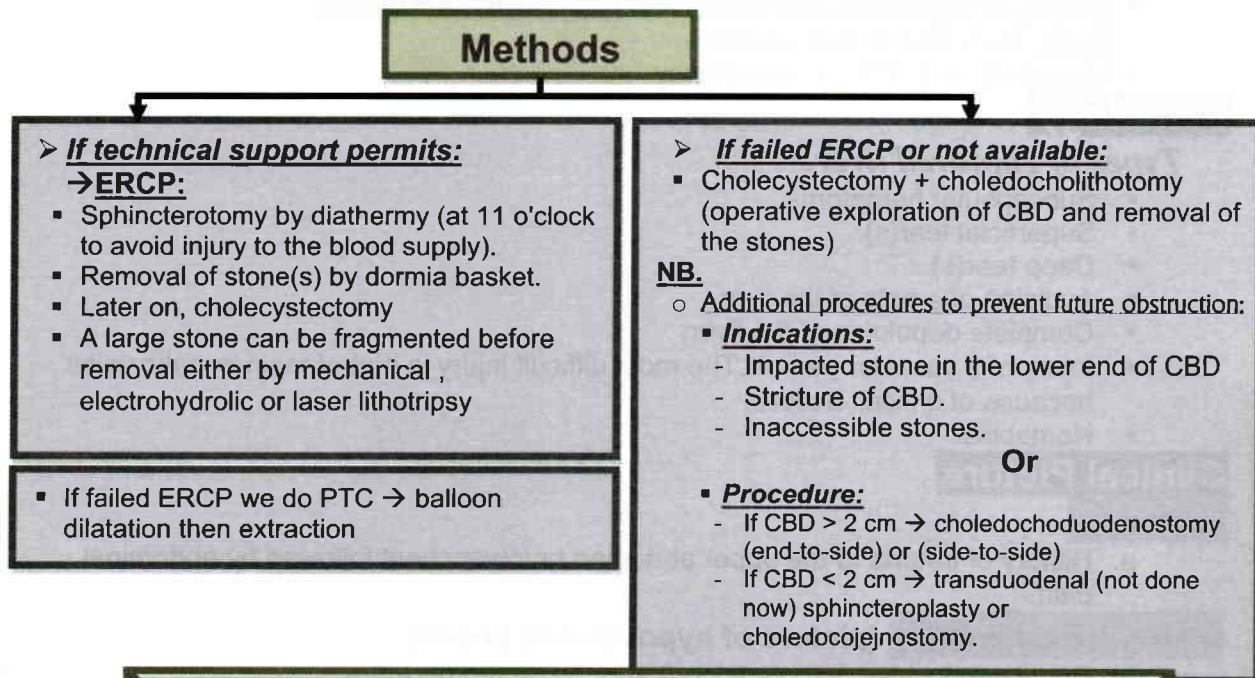
- Abdominal U/S: (*U/S is less sensitive to lower part of CBD*) & the first to be done
 - CBD stones are suggested by CBD diameter > 8 cm on U/S.
 - Dilated intrahepatic biliary radicals.
 - Chronically inflamed GB with stones.
- ERCP: (*indicated in OJ with suspected lesion in lower end of the CBD*)
- PTC: (*indicated in obstructive jaundice with suspected lesion in upper end of CBD*)
- MRCP: (good diagnostic value but not therapeutic)
- In malignant OJ: as above +
 1. Tumor markers: CEA, POFA, PCAA.
 2. Barium meal.

Treatment

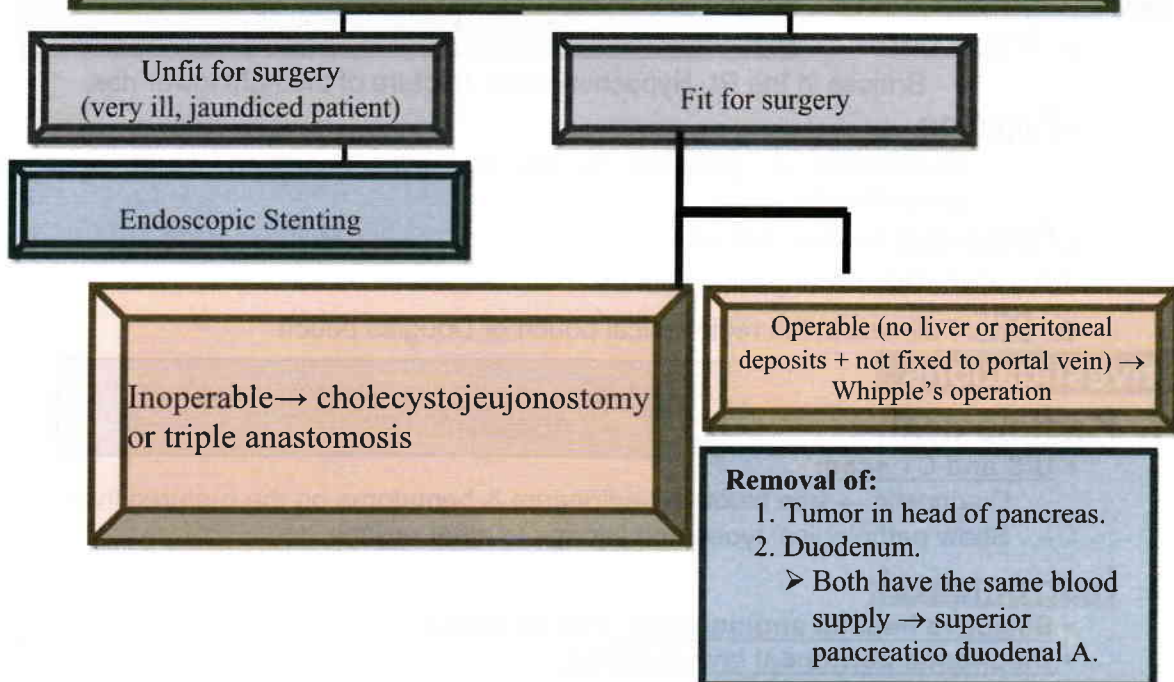
A. Preoperative Preparations: (glucose, Vit.k, Ca glucometer and antibiotics)

B. Definitive Treatment

🔥 **In case of Calcular OJ**



According to the general condition of the patient



LIVER

Liver Trauma

Etiology

- **Closed:** Direct trauma, Indirect trauma or Spontaneous rupture
- **Open:** Gun-shot or stab wound.
- **Iatrogenic:** e.g. PTC or liver biopsy.

Pathology

Types of ruptured liver:

- Subcapsular hematoma.
- Superficial tear(s).
- Deep tear(s).
- Avulsion of a pole of the liver.
- Complete depulping of the liver.
- Injury of a vascular pedicle. The most difficult injury is that of main hepatic veins because of difficult access.
- Hemobilia.

Clinical Picture

History

- a. History of trauma to the upper abdomen or lower chest followed by abdominal pain.

General Examination (picture of hypovolemic shock)

- a) Rapid weak pulse, hypotension & subnormal temperature.
- b) Cold extremities & pallor.

Local Examination

We have to exclude other associated injuries of the chest, abdomen & head

- a) **Inspection:**
 - Bruises in the Rt. Hypochondrium, fracture of the right lower ribs.
- b) **Palpation:**
 - Tenderness & guarding in the Rt. hypochondrium. Later becomes generalized.
- c) **Percussion:** Shifting dullness.
- d) **Auscultation:** ↓ intestinal sounds.
- e) **DRE:** Fullness in the rectovesical pouch or Douglas pouch.

Investigations

▪ **The most important diagnostic tools are**[□]:
- Abdominal U/S & CT.

1. Radiological:

- **U/S and CT scan:**
 - Diagnostic → free blood in peritoneum & hematoma on the ruptured liver.
 - Show pathological types and injuries to other organs.

2. Instrumental:

- **Selective hepatic angiography:** may be helpful.
- **Diagnostic peritoneal lavage (DPL).**

3. Laboratory investigations:

- KFTs, LFTs, FBS, electrolytes, CBC.

Treatment

- ▶ Management of polytraumatized patient (advanced trauma life support)

1. Prehospital management ABCD

2. Hospital management

- a- Primary survey: ABCD
- b- Secondary survey:

3. Preoperative Preparation

- Blood transfusion and morphia.

4. Immediate Laparotomy

- The priority is to arrest bleeding:
 - If the bleeding stops by the time of exploration, just put a drain
 - We control the liver hemorrhage by a combination of temporary packing of the bleeding area and application of the Pringle's manoeuvre[☐] for 20 minutes+ fresh frozen plasma.
- Dealing with different types of injury
 - Suturing liver tears should be avoided (whenever possible) as it may cause a hematoma → infection or hemobilia.
 - However: deeply placed mattress sutures supported by a pad of omentum is recommended.
 - If there is hematoma: we ligate damaged vessels and ducts and excise dead tissues.
 - Firm packing of difficult and inaccessible areas eg: with hepatic veins it may be the only method for temporary arrest of bleeding
- Multiple intraperitoneal drains: to avoid collections of blood and bile.
- Prophylactic antibiotics.

Hydatid Disease of the Liver

Cause

- Infection by larval stage of **Echinococcus granulosus**.
- **Echinococcus multilocularis** (rare) → malignant hydatid disease.

Human Affection

1. As accidental **intermediate host**[☐] (blind host).
2. infected by:
 - Ingestion of eggs with dirty vegetables
 - Playing with dogs.

Pathology of Hydatid Cyst

Site

- Common in the Rt. lobe[☐].

Number

- Common to be solitary but may be multiple

Layers of the Cyst (Double layer cyst wall)

- 1- Adventitia (pericyst)
 - Fibrous layer formed by liver
- 2- Laminated membrane (ectocyst)
 - Formed by the parasite from germinal layer Separated from adventitia by plane of cleavage, but adherent infections
- 3- Germinal layer (endo-cyst):
 - It gives:
 1. Scolices (head of future worms).
 2. Brood capsule → Collection of scolices.
 3. Daughter cyst: May be formed outside the cyst or inside it.
- 4- Hydatid fluid: i
 - Colourless and clear but may be yellowish if the cyst communicates with the bile duct

Clinical Picture

Type of Patient

1. Common in people dealing with sheep.
2. Common in certain countries as Saudi Arabia, Iraq, Libya & Sudan.

Symptoms

1. **Asymptomatic** (the most common[□]).
2. Chronic pain in Rt. upper quadrant.
3. **Swelling in the** upper abdomen.

Signs

1. Swelling has the criteria of liver swelling.
2. Hydatid thrill might be felt on percussion (Due to vibration of daughter cyst in fluid) in 70% of cases
3. Hepatomegaly.

Fate & Complications

- The majority of cases, the cyst **gradually enlarges**.
- In others, the **parasite dies** and the **cyst is calcified**.
- Rarely, the cyst undergoes one of the following complications:
 1. Rupture in[□]:
 - *Biliary tree* → obstructive jaundice (*the commonest*).
 - *Peritoneum* → severe anaphylaxis, urticaria (generalized hydatidosis) and diffuse peritonitis.
 - *Hepatic veins* → systemic affection especially lung.
 2. Spread to other organs: (lung, brain & bone)
 - More in E. multilocularis.
 3. Secondary infection (10-15%) Pyogenic liver abscess → painful.
 4. Pressure: On the surrounding structures.
 5. Calcification[□].
 6. Hemorrhage.

Investigations

A. For diagnosis:

I. Laboratory:

▪ Serological:

1. Complement fixation test → + ve in 95% of cases.
2. Haemagglutination test → + ve in 95% of cases.

3. Casoni intra-dermal test[□] → +ve in 70% of cases (Not widely used now due to its low sensitivity and specificity).

- **CBC:** Eosinophilia.
- **LFTs:** ↑ direct bilirubin & alkaline phosphatase (if jaundiced).

2. Radiological:

- **U/S & CT scan:** can detect size site and number of cysts.
- **Plain X-ray chest & abdomen:**
 - Elevated Rt. copula of the diaphragm.
 - Rt. Sided pleural effusion (± lung affection)
 - Calcified cyst.

B. For complications:

- **ERCP:**
 - Indication: if the cyst ruptures into the bile duct causing obstructive jaundice

Treatment

Mainly Surgical

Medical TTT + surgery + medical TTT

A. Sterilization of cyst:

Injection of scolicial material in the cyst (Hypertonic saline 25% or Povidone iodine {betadine}) by Double-way syringe or Hydatid cone.

B. Removal of cyst:

1. Enucleation (best treatment):

- through the plane of cleavage between the adventitia & laminated membrane.

2. Removal in block: in absence of plane of cleavage.

3. Hepatic lobectomy: if multiple cysts

+ Obliteration of the cavity:

- Omentoplasty (by pedicle omentum).

Medical Treatment

1. Indications:

1. Disseminated disease.
2. Preoperatively to avoid complications during surgery (4 days before operation).
3. Post operatively to kill any spilled scolices and prevents recurrence for 1-3 months.
4. Small asymptomatic cyst.
5. Elderly patients.

2. Types:

I- **Mebendazole** Or **Albendazole** (less toxic)

II- **Hydrocortisone:** by injection for a day before, during and after any surgical procedure.

Treatment of Complications

- a- Obstructive jaundice → explores CBD surgically or by endoscope.
- b- Infected cyst (abscess) → drain & antibiotics.

Amoebic Liver Abscess

(Tropical abscess or dysenteric abscess)

Causative Organism

- **Entamoeba histolytica** (As a complication of amoebic colitis).

Mode of Infection

Cysts are the infective form

1. **Ingestion** of food or drinks contaminated by **Entamoeba cyst**.
2. Cyst hatches in intestine → trophozoite form releasing proteolytic enzymes.

Pathology

Site

- Rt. Lobe is affected more than Lt. lobe[☐]:
- The posterosuperior segment of the Rt. lobe of liver is the commonest site as the segmental branch of Rt. portal vein (to this segment) is in direct continuity with Rt. portal vein.

Number

- Solitary in 70% of cases.

Pus

- Usually sterile with brown chocolate colour and sometimes it is pinkish (anchovy sauce). The wall is shaggy and harbors amoeba.

Pathogenesis

Amoebic Hepatitis

- It is liquefactive necrosis of liver tissue.

➤ Fate:

- Healing by medical treatment. OR
- Coalescence together to form an abscess.

Amoebic Liver Abscess

- It is **not a true abscess**[☐] as there is no pyogenic membrane.

➤ Fate:

- **Complete Resolution:** is the rule.
- **Chronicity:** the abscess becomes surrounded by thick fibrous layer.
- **Complications:**
 1. Rupture towards:
 - Peritoneum → **peritonitis**.
 - Sub-phrenic space → **Pleura**[☐] → Lung → coughing of chocolate sputum (commonest).
 - **Pericardium** (amebic pericarditis & myocarditis).
 2. **Secondary infection:** forming pyogenic abscess.
 3. **Pressure effect:** devitalizing adjacent liver cells.
 4. **Calcification.**

Clinical Picture**Symptoms**▪ **General:**

Severe malaise, loss of weight, fever, profuse sweating, rigors, Nausea & vomiting, cough of anchovy like material

▪ **Local: Pain**

1. Severe, in the Rt. hypochondrium.
2. Radiates to the Rt. Shoulder.

Signs▪ **General:**

Low grade fever (*if high* → *2nd infection* → *Pyogenic abscess*),
Pallor & toxic look. + Rt. sided pleural effusion.

▪ **Local:**

1. Inspection: Rigidity & pitting edema over the lower right ribs.
2. Palpation: Tender hepatomegaly.
3. Percussion: Rt lower Inter-costal tenderness.

Investigations▪ The most important diagnostic tools are:

- 1- U/S.
- 2- Metronidazole test.
- 3- Isolation of the parasite from the liver or stool.
- 4- Stool analysis in non-endemic areas.

1. **Laboratory:**

▪ **Serological:** CFT, IHA. Gel diffusion precipitative test is +ve in 95% of amoebic abscesses.

▪ **Stools analysis:**

- Useful in non endemic areas.

▪ **Therapeutic test:**

- Toxic symptoms will improve on **metronidazole** as a therapeutic agent (diagnostic & therapeutic).

2. **Radiological:**

▪ **U/S & CT:** Detects number, site & size of abscess.

Treatment**Medical Treatment**

→ highly successful

▪ **Metronidazole:** Drug of choice → 800 mg 3 times daily for 7 – 10 days.

Surgical Treatment

1-Aspiration guided U/S: (under cover of Metronidazole using wide bore needle under local anesthesia)

Indications:

1. Resistant after medical treatment (within 72 hours).
2. Large abscess.
3. Rupture.
4. Patient is very toxic.

2-Open drainage

➤ Indications:

1. Failure of aspiration (thick contents, multilocular).
2. Rapid refilling, or rupture leading to peritonitis.
3. Secondary infection.
4. Abscess in the Lt. lobe (for fear of rupture in pericardium).

➤ Technique:

- Rt. lobe → extra-pleural extra-peritoneal through bed of 12th rib.
- Lt. Lobe → Midline.

Liver Tumors

Benign Tumors

- 1- Adenoma.
- 2- Hemangioma (most common benign liver tumor).
- 3- Focal nodular hyperplasia.
- 4- Hamartoma.

Malignant Tumors

1- Primary tumors:

- Hepatocellular carcinoma (HCC).
- Cholangiocarcinoma.
- Cholangiohepatoma.
- Hepatoblastoma (D.D. Neuroblastoma, Wilm's tumor).

2- Secondary tumors: Metastasis.

Hepatocellular carcinoma (hepatoma)

Incidence

-More in old males.

Predisposing Factors : (90% of cases are related to HCV, HBV & liver cirrhosis)

1. Hepatitis B & C.
2. Liver cirrhosis of any cause.
3. Carcinogenic material: as Aflatoxins
4. In children: Biliary atresia, α 1-antitrypsin deficiency & Galactosemia.
5. Adenoma: (Rare).

Pathology

Macroscopic: infiltrating mass edge with areas of Hge & necrosis

It may be: **Massive form, Nodular form or diffuse form**

Microscopic: -Malignant hepatocytes (adenocarcinoma) with pleomorphism,

↑ Mitotic figures & ↑ N/C ratio.

-High vascularity derived from **hepatic artery** (not from the portal vein).

Fibrolamellar type: It is a variety of massive form affects non-cirrhotic young females with Better prognosis & -ve alpha fetoprotein.

Spread

Direct

- To stomach, colon, kidney & diaphragm.

Lymphatic

- To LNs in porta hepatis → celiac LNs → thoracic duct → Lt. supra-clavicular LNs "Virchow's LN."

Blood

- To Lung & bones.

Clinical Picture of Liver Tumors

- Rapid deterioration of health of a patient with liver cirrhosis → Suspect hepatoma ☑.

Frank (Hepatic) Presentation

- **Symptoms:**
 1. History of chronic hepatitis & liver cirrhosis.
 2. Exaggeration of pre-existing hepatic condition:
 - a. Jaundice.
 - b. LCF → hepatic encephalopathy.
 - c. Portal hypertension → hematemesis & resistant ascites.
 3. Swelling → (Late) localized to liver.
 4. Pain → (Late) dull aching in upper abdomen.
N.B. Hepatocellular carcinoma in black Africans may resemble amoebic hepatitis with pain and fever.
- **Signs:**
 1. **General:**
 - a. Jaundice, fever and cachexia.
 - b. Enlarged supra-clavicular L.N. (Lt. & Rt.).
 2. **Local:**
 1. Liver is enlarged, irregular & tender.
 2. Localized tender mass may be felt (in 50%).
 3. Ascites.
 4. Occasionally murmur over the tumor (Mamoun sign[□]).

Occult (Extra-hepatic) Presentation

- **Non-metastatic(Paramalignant syndrome[□]):**
 1. Hypoglycemia due to Insulin-Like Polypeptide(ILP).
 2. Polycythemia (erythropoietin).
 3. Hypercalcemia & pathological fracture (PTH like)
 4. Cushing like (ACTH) .
 5. Hypotension (VD) or Hypertension (ACTH)
 6. Fever (FUO).

Investigations**I. For diagnosis:****-U/S, CT or MRI:**

- Detect the site and extent of tumor

-Tumor markers:

1. Carboxy prothrombin:
 - More specific.
2. Alpha fetoprotein[□]: (elevated in 55-95% of patients)
 - Useful tumor marker for follow up although its low sensitivity..
 - Level above 2000 ng/dl is diagnostic of HCC

-Hepatic angiography: (through hepatic artery)

- Shows characteristic high vascularity.

-Percutaneous guided needle biopsy (by U / S or CT):(Provided that PT is normal)

- Point of controversy.
- Done only in inoperable cases before chemotherapy.

-Laparoscopy: can give us direct biopsy.

2. For staging:

- Chest X-ray and CT.....

3. For preoperative preparation:

- LFTs, KFTs, CBC, FBS...

Treatment**Operable Cases**

- The ideal treatment for a tumor that is localized to one liver lobe is to resect it rather than transplantation provided that the liver is non-cirrhotic.
- The presence of cirrhosis (this indicate transplantation[□]) in most cases is a relative contraindication due to:
 - 1- Bleeding tendency.
 - 2- Poor function of the remaining part of liver.
 - 3- Diminished capacity of the liver cells to regenerate.

Inoperable Cases

1. Chemotherapy
2. Ligation of feeding artery.
3. Chemoembolization
4. Cryosurgery.
5. Percutaneous ethanol injection in the tumor
6. Thermal ablation by:
7. Monoclonal antibody therapy.

Liver Metastasis

- It is the **most common** malignant tumor of the liver (20 times more common than 1^{ry} malignancies) [□].
- **The commonest primary sites are:**
 - a. Stomach, breast, kidney.
 - b. Colorectal, lung.
- **The metastasis reach the liver via:**
 - a. **Portal vein:** (The commonest route)
 - b. **Hepatic artery**
 - c. **Lymphatics → breast.**
 - d. **Direct extension:** → E.g. tumours of G.B., stomach & colon.

Pathology

- They are multiple, umbilicated nodules due to central necrosis.
- They are usually adenocarcinomas
- Over 90% of cases have tumor deposits in other organs

Clinical Picture

- Picture of primary tumor +
- Hepatomegaly, ascites and jaundice.

Investigations

1. **Laboratory:**
 - **Tumor markers.**
2. **Radiological:**
 - **U/S, CT or MRI and PET-scan:**
- Are diagnostic and show the number and sites of metastases.

Treatment

Treatment of non-resectable tumors

1. **Chemotherapy:**
2. **Monoclonal antibodies.**
4. **Sectorial Portal vein emolization** (atrophy of the affected segment).

Surgical Resection

- **Prerequisites:**
 - 1- Completely resectable 1^{ry} tumor.
 - 2- Solitary liver metastasis or multiple (<4) confined to one surgical lobe
 - 3- Liver is the only affected organ with metastasis.

Portal Hypertension

Definition

- Portal hypertension is present when portal vein pressure exceeds 20 mmHg (25-30 cmH₂O).

Etiology

I- Prehepatic Causes:

- 1. Congenital narrowing or atresia:** of portal vein.
- 2. Portal vein thrombosis** secondary to neonatal umbilical sepsis, polycythemia.
- 3. Invasion by malignancy:** e.g. HCC, pancreatic carcinoma.

II- Intrahepatic Causes:

- 1- Pre-sinusoidal:** bilharzial fibrosis, congenital fibrosis.
- 2- Sinusoidal:** liver cirrhosis.
- 3- Post-sinusoidal:** veno-occlusive disease.

III- Suprahepatic Causes:

- 1- Budd-chiari syndrome (occlusion at hepatic vein[☑]).
- 2- High IVC obstruction.
- 3- Constrictive pericarditis and pericardial effusion.

Pathology

I. Portosystemic Anastomosis:

The important sites of these collaterals are:

Site	Between	
	Portal circulation	Systemic circulation
1- Lower end of esophagus and the fundus of the stomach (esophageal & fundic varices) [☑]	Esophageal veins from Left gastric vein (coronary vein) & short gastric veins	Esophageal veins from Azygous & hemiazygous veins
2- Around umbilicus (Caput Medusae) [☑]	Paraumbilical vein	Superior and inferior Epigastric veins
3- Lower end of the rectum and Anal canal (anorectal varices) [☑]	Superior rectal veins	Middle & inferior rectal veins
4- Others ➔ Retroperitoneal space	Superior and inferior mesenteric veins	Posterior abdominal wall veins and subdiaphragmatic veins

2. Splenomegaly:

- **Causes:** RES hyperplasia and congestive splenomegaly.
- **Results:**
 - A. Hypersplenism.
 - B. Recurrent attacks of splenic infarctions.

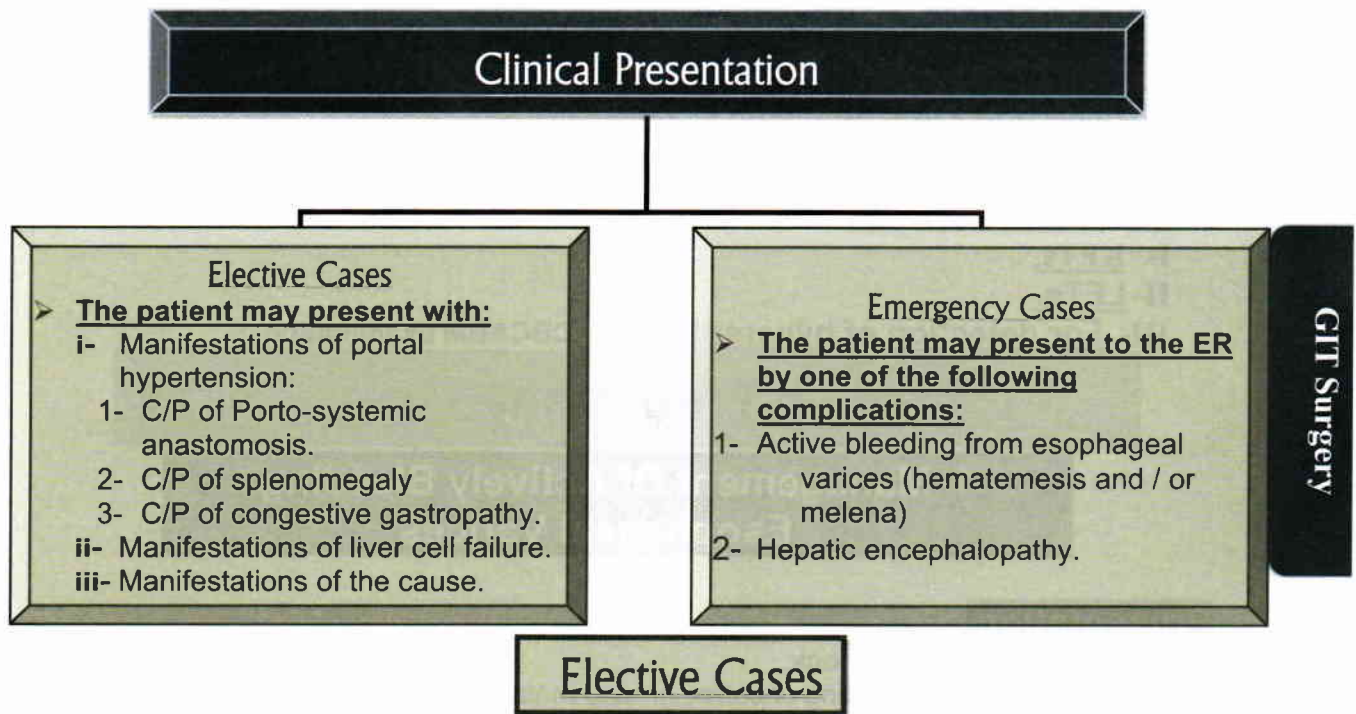
3. Ascites:

- **Causes:** (It is multifactorial): Hypoalbuminemia, portal HTN, salt and water retention and lymphorrhea.

4. Congestion of the Whole GIT: congestive gastropathy & enteropathy.

5. Liver: (according to the cause of portal hypertension)

1. Prehepatic causes: normal
2. Post-hepatic causes: enlarged, soft, tender.
3. Hepatic causes: hepatomegaly then shrank.



I- Manifestations of portal hypertension:

- 1- Clinical picture of Porto-systemic anastomosis:
 - a. Esophageal varices. Hematemesis and melena.
 - b. Caput medusa. Dilated veins around the umbilicus.
 - c. Anorectal varices (rare).
- 2- Clinical picture of Splenomegaly:
 - Symptoms: abdominal pain and enlargement.
 - Signs: Firm mass in Lt. hypochondria with preserved notch
- 3- Clinical picture of congestive gastropathy:
 - Anorexia, dyspepsia, indigestion and malabsorption.

Investigations

1- For diagnosis:

- **Fiberoptic upper endoscope:** both diagnostic & therapeutic.
- **Abdominal U/S:** It show Liver Cirrhosis, splenomegaly, ascites, portal vein diameter (>14mm in case of portal HTN), hepatoma.
- **Barium swallow** (not used now): show varices as multiple, smooth, rounded filling defects.
- **Duplex scan:** Pressure & patency of portal vein.

2- For the cause:

- **Sigmoidoscopy:** To visualize bilharzial lesions and take biopsy snips
- **Hepatitis markers.**
- **Urine and stool analysis:** for bilharzial ova
- **Liver biopsy after assessment of PT and PC.**

3- For complications:

I- KFTs.

II- LFTs.

III- For detection of hypersplenism: CBC&BM examination.

Treatment

1- Management Of Actively Bleeding Esophageal Varices

Presentation

1. **Variable degrees of shock.**
2. **Hematemesis** → Vomiting of coffee ground material (acid hematin formation) due to bleeding above the ligament of Trietz.
(It may be red blood if severe bleeding)
3. **Melena** → Passage of soft tarry offensive stool
(It may be red blood if severe bleeding due to GI hurry)
4. **Picture of the cause:**
 - a- Bilharziasis → history of bilharziasis, anemia, easy fatigability & HSM
 - b- Cirrhosis → Jaundice, encephalopathy, shrunken liver & splenomegaly.

Investigation of choice

-Upper GIT endoscopy.

Treatment

A- Hospitalization:

Even minor hematemesis may be followed by massive bleeding.

B- Resuscitation and monitoring:

- **IV line:** IV fluids & blood transfusion.
- **Ryle.**

- Catheter: to monitor urine output.
- Monitoring of: BP, pulse.
- Correct coagulopathy by:
 - Fresh blood (more platelet & coagulation factors & less ammonia & K).
 - Fresh frozen plasma.
 - IV vitamin K.
 - Hemostatics e.g. cyclokapron, Dicynon.
- Sedatives:
 - Avoid morphine and pethidine due to liver affection (we can use paraldehyde or chloral hydrate).

C- Prevent encephalopathy:

- Aim: evacuate blood from GIT & prevents its fermentation.
- Method:
 - Gastric lavage through Ryle tube by cold saline every hour.
 - Enema every 2 hrs.
 - Enteral antibiotics e.g. neomycin 1 gm. / 6 hrs. or Metronidazole 250 mg / tads
 - Lactulose (10 – 30 ml tads orally). It is fermented to lactic acid → combines with ammonia and has mild laxative effect.
 - Avoid hypokalemia and sedatives.

D- Stop bleeding

i. Urgent upper GIT endoscopy:

- Confirm the source of bleeding (**diagnostic**).
- For injection Sclerotherapy or band ligation (**therapeutic**).
 - Injection Sclerotherapy[□]: (most effective treatment that preserve hepatic portal perfusion)
 - Method:
 - ▶ Intravariceal:
 - 5 ml ethanolamine oleate (in esophageal varices).
 - Histoacryl blue "Bucrylate" (in gastric varices).
 - ▶ Paravariceal: sub-mucosal injection of sclerosant.
 - 1/2 ml of aethoxysclerol is injected to produce perivascular fibrosis.
 - Result:
 - Stops bleeding in 90% of cases.
 - Band ligation:
 - Encircle each varix by a tight band → thrombosis of the veins.
 - As effective as sclerotherapy with fewer side effects.

ii. Drugs:

- Vasopressin
 - Action:
 - vasoconstriction of splanchnic arterioles & venules → ↓ portal venous pressure (portal vein has no smooth muscle)
- Glypressin (vasopressin + Glycine):
 - Has more prolonged action & fewer side effects.
- Somatostatin: (tetradecapeptide).
 - Stronger action and fewer side effects

➤ **Propranolol:**

- Lowers portal pressure, reduce liver blood flow and cardiac output.
- Prevent recurrent variceal bleeding in compensated cirrhotic patients.

iii. **Balloon tamponade by:**

Linton-Nachlas tube, Sengstaken-Blakmore or Minnesota tube.

• **Disadvantages**

- Discomfort sensation.
- The patient can not swallow his saliva.
- Esophageal rupture or pressure necrosis[☑].
- Rebleeding in 60 – 80 % of patients[☑]
- Rupture of gastric balloon → suffocation and death.

IV. Transjugular intrahepatic porto-systemic shunt(TIPSS):

- It is the recommended method if all the previous methods failed to stop bleeding..

2- Silent Varices

- Prevention of bleeding from silent varices is important since the lifetime risk of a first-time variceal bleed in a cirrhotic patient is 30% and carries a mortality rate of up to 50%.
- Primary prevention with a non-selective beta-blocker (propranolol or nadolol) is started if large varices are found, especially if they have stigmata of bleeding (red streaks on the variceal surface).

3- Patients with Past History of Bleeding Esophageal Varices

Recently:

- Whatever the category, repeated sclerotherapy is indicated until the varices are obliterated.
- **If Sclerotherapy fail:**
 - Child A: Porto-systemic shunt or devascularization.
 - Child B: Devascularization only.
 - Child C: Not fit for surgery, the only hope is liver transplantation.

More about Surgical treatment of portal hypertension

Shunt Surgery

A. Total Shunt (shunt whole portal blood into the systemic circulation)

1- Porto-caval Shunt:

- The distal end of the portal vein is divided and transected while the proximal end is anastomosed to the anterior surface of IVC.

2- Meso-caval shunt (Drapanas):

- Idea: Insert a jump graft between SMV & IVC.

3- Proximal spleno-renal shunt:

- Technique:
 - Splenectomy is performed then the proximal end of splenic vein is anastomosed to Lt. renal vein.

B. Selective Shunt

1. Distal lieno-renal shunt (Warren's operation):

- Idea: Selective decompression of gastro-esophageal varices without disturbing mesenteric or portal venous flow.
- Technique:
 - The Rt. & Lt. gastric vessels are ligated.
 - The splenic vein is mobilized; its proximal end is ligated, while the distal end is anastomosed to the Lt. renal vein.
 - The short gastric veins are preserved and will decompress the lower end of esophagus.

Devascularization Procedures

(Porto azygos disconnection)

- They are non-shunting procedures used to treat gastro-esophageal hemorrhage.
- Best treatment of variceal bleeding 2ry to portal vein splenic & S.M.V. thrombosis.

➤ Indications of devascularization operations:

- Failure of sclerotherapy in patients with good general condition.

1- Hassab-Khairi Operation:

➤ Advantages:

- No encephalopathy.
- Portal flow is intact.
- Low incidence of rebleeding.

2- Esophageal Transection & Anastomosis:

- The purpose of this operation is interruption of submucosal venous plexuses which is still intact following porto azygos disconnection.
- It is simple and easy and can be added to porto azygos disconnection.

Liver transplantation

- Is indicated for patients with end-stage liver disease.
- It will correct the problem of the varices and ascites.

SPLEEN

Rupture of the Spleen

- It is the most common organ to be injured in blunt abdominal trauma[☑].

Predisposing Factors

- Splenic enlargement: which makes it more liable to trauma.
- Diseases of the spleen: e.g. malaria & typhoid, which make it soft.

Etiology

➤ Trauma, which may be:

- Closed:
 - Direct trauma (blunt trauma) e.g. car accident or falling from a height.
 - Indirect trauma: fracture ribs.
 - Spontaneous rupture: with pathological spleen.
- Open:
 - Gun-shot wounds.
 - Stab wounds.
 - Iatrogenic: e.g. gastrectomy.

Pathology

➤ Types of ruptured spleen:

- Subcapsular hematoma.
- Superficial tear(s).
- Deep tear(s).
- Avulsion of a pole of the spleen.
- Complete depulping of the spleen.
- Injury of a vascular pedicle.

Complications

- Hemorrhage: internal or external.
- Splenic cyst: may follow a perisplenic hematoma.
- Associated abdominal or thoracic injuries.

Clinical Picture

History

- History of trauma to the upper abdomen or lower chest followed by abdominal pain.

Examination

There are three types:

1- Classic type (commonest):

It passes into 3 stages:

- **Stage of shock.**
- **Stage of recovery** from shock (Lucid interval) d.t. arrest of bleeding from ↓BP.
- **Stage of internal hemorrhage** due to rise of blood pressure after resuscitation.

⇒ **General examination→(signs of shock)**

- Rapid weak pulse, hypotension.
- Subnormal temperature, cold extremities & pallor.
- Decrease in urine output despite adequate fluid replacement.

⇒ **Local examination:**

- Inspection:
 - Bruises in the Lt. Hypochondrium.
 - Fracture of the Lt. Lower ribs.
- Palpation: Tenderness, RT& guarding in the Lt. Hypochondrium.
- Percussion: shifting dullness.
- Auscultation: ↓ intestinal sounds.
- DRE: tenderness, fullness in the rectovesical pouch.

⇒ **Special signs[□]:**

- **Kehr's sign:** referred pain to the Lt. Shoulder ± hyperthesia from diaphragmatic irritation by blood, especially with Trendelenberg position.
- **Cullen's sign (late):** bluish discoloration around the umbilicus due to absorption of the intraperitoneal blood by lymphatics around the umbilicus.
- **Balance's sign (late & pathognomonic):**(+ve in 25%of cases)
 - Shifting dullness on the Rt. side from the free blood in the peritoneum.
 - Fixed dullness on the Lt. side due to clots & retroperitoneal hematoma.

2- Fatal type:

- Due to tearing of the splenic vessels or complete avulsion of the splenic pedicle.
- The patient is severely shocked & usually dies within a short period.
- Usually associated with other visceral injuries.

3- Delayed type:

- The initial shock is followed by long lucid interval up to 15 days, after which the patient presents with features of internal hemorrhage. This delay is due to:
 - Formation of subcapsular hematoma which may be ruptured later.
 - The greater omentum seals the splenic tear then retracts later on.
 - Blood clots seal the splenic vessels then dislodges when Bl. Pr. rises.

Investigations▪ **The most important diagnostic tools are[□]:**

- Abdominal U / S & CT scan.

1. Radiological:

- **Abdominal U / S & CT scan(most useful):**
 - Are diagnostic & show pathological type of rupture.
- **Plain x-ray:**
 - Fracture in the lower left ribs.
 - Elevated left copula of the diaphragm[□].

2. Instrumental:

- **Diagnostic peritoneal lavage[□] (greatly replaced by US and CT):**
 - Intra-peritoneal hemorrhage is diagnosed if there is > 100,000 RBCs/ml.

3. Laboratory:

- CBC, KFTs, FBS, electrolytes ...

If the patient is in severe shock there is no time for investigations and the surgeon should depend on clinical findings for surgery.

Treatment

Pre-operative Preparation

⇒ Blood transfusion, IV fluids, morphia, vit K, warmth.....

Surgery

- ⇒ Adult → immediate laparotomy & Splenectomy.
- ⇒ Children → splenic preservation by:
 - Splenorrhaphy: suturing of small tears.
 - Splenic artery ligation (occasionally indicated with splenorrhaphy)
 - Splenic embolization by gel foam.
 - Partial Splenectomy: if avulsed one pole.
 - Splenic mesh wrap: if avulsed whole spleen.

Precautions should be done if Splenectomy is indicated before five years old:

- **Preoperative:** vaccination against the capsulated organisms[□] (pneumococci, meningococci, H. influenza) to avoid OPSI (overwhelming post splenectomy infection)
- **Pneumovax** : is given every 5 years until the age of 18.
- **Postoperative:** long acting penicillin.

PANCREAS

Acute Pancreatitis

Definition

- Acute inflame. of the pancreas with release of digestive enzymes and autodigestion of the pancreas.

Etiology

1. Bile Duct Stones (COMMONEST → 50%)
2. Excess Alcohol Intake (35%)
3. Idiopathic Pancreatitis (20%).
4. Postoperative (Iatrogenic) Pancreatitis: e.g. ERCP, after splenectomy or gastrectomy.
5. Periapillary carcinoma.
6. Other rare causes: Hypercalcemia, hyperlipidemia....

Pathology

- In acute pancreatitis there is premature activation of the pancreatic digestive enzymes. This leads to auto digestion of the gland.
- The peritoneum is full of abundant protein rich exudates.
- The enzyme lipase splits fat in the omentum and peritoneum producing glycerol and fatty acids, this acid binds with calcium forming insoluble calcium soap which manifests as white spots of fat necrosis.
- The proteolytic activity of the liberated enzymes produces vaso active kinins and other chemical mediators which are responsible for the hemodynamic and respiratory effects (systemic inflammatory response syndrome ; SIRS)

Clinical Picture

Symptoms

A-General: FAHM

B-Local:

- 1- **Severe epigastric pain radiating to the back**, relieved in early cases by the prayer's position.
- 2- **Repeated vomiting**.
- 3- **Hematemesis & melena**: due to stress ulcer.
- 4- **History of the cause**

Signs

A. General:

1. **↑temp. & tachycardia**.
2. **Signs of complications (multiorgan failure) as:** Cyanosis, shock or jaundice.

B. Local:

1. Inspection:

- 1- Epigastric rigidity.
- 2- In late cases:

Cullen's sign → Bluish discoloration around umbilicus.

Grey Turner's sign → Bluish discoloration of both flanks.

2. **Palpation:** Epigastric tenderness

3. **Percussion:** Shifting dullness.
4. **Auscultation:** ↓ Intestinal sounds.

DD *Of acute upper abdominal pain*

- 1) **Acute cholecystitis** → epigastric pain radiating to Rt. shoulder & back (Boa's & leak signs).
- 2) **Acutely perforated PU** → Peptic dyspepsia.
- 3) **High small intestinal obstruction** → absolute constipation, ↑ intestinal sounds & on X-Ray → multiple fluid levels.
- 4) **Dissecting aortic aneurysm.**
- 5) **Acute mesenteric vascular occlusion (MVO)**
- 6) **Acute inferior wall myocardial infarction (MI)**

Complications

A. Systemic complications: “MOF” *multiorgan failure*

1. **Shock:** hypovolemic or neurogenic
2. **ARDS.**
3. **Renal failure:** d.t hypovolemia or as a part of MOF.
4. **Pleural & pericardial effusion**
5. **DIC:** d.t consumption of coagulation factors.
6. **Tetany:** due to hypocalcaemia.
7. **Lt. sectorial portal hypertension** (splenic vein thrombosis).

B. Local complications:

1. **Pseudo pancreatic cyst**
2. **Pancreatic abscess.**
3. **Chronic pancreatitis.**

Investigations

1. For diagnosis:

1. **Serum Amylase:** (*nonspecific*):
 - a. Elevated within few hours & drops after 5 days.
 - b. Diagnostic level: > 1000 somogyi units.
2. **Serum Lipase (specific):** increased
3. **CT scan:** enlarged pancreas, tissue necrosis, & fluid collection in peritoneum.
4. **Plain X-Ray of the abdomen (usually not done):** Colon cut off sign or Sentinel loop ileus sign.
5. **ECG & cardiac enzymes to exclude MI**

2. For the cause:

- ▣ **Abdominal U/S:** May show gall stones.

3. For complications:

1. **CBC:** leukocytosis
2. **LFTs:** ↑ bilirubin
3. **KFTs:** Renal failure.
4. **Fasting blood sugar:** ↑ due to ↓ insulin
5. **Serum Ca:** ↓

Ranson's criteria → Bad Prognostic criteria

On Admission			Within the First 48 hrs.		
Age	>	55 years.	Base Deficit	>	4 mEq/L
Fasting blood sugar	>	200 mg%	Estimated fluid sequestration	>	6 L
WBCs count	>	16 000/mm ³	Serum Ca	<	8 mg%.
Serum LDH	>	350 IU/ml ²	BUN	>	5 mg%.
SGOT	>	250 U%.	Arterial O₂	<	60 mmHg

Treatment**A. Conservative(R regimen):**

1. Relief of pain: by pethidine or atropine(morphia not used)
2. Replacement of the lost fluids: by IV fluids or blood
3. Rest of the pancreas and bowel: NG suction and NPO+somatostatin.
4. Respiratory support: **O₂ mask or mechanical ventilation**
5. Resistance of Infection: by antibiotic e.g. 3rd generation cephalosporins.

B. Surgical:**- Indications of Surgery:**

- doubtful diagnosis (→ drain & close the abdomen).
- in late cases to remove the necrotic tissue.

-Complications e.g.:

- Pseudo pancreatic cyst → Cyst gastrostomy or Cystojejunostomy.

C. Treatment of the cause:

- e.g. CBD stones

Pancreatic pseudo cyst**Pathology**

- **Nature** : this is a collection of pancreatic secretion and inflammatory exudates within a lining of inflammatory tissue rather than epithelium.
- **Etiology**: -develops in 10% of cases of acute pancreatitis.
-the next common cause is pancreatic trauma
- **Site** : commonly in the lesser sac between the pancreas and the stomach
- **Complications** : infection ,Hemorrhage & rupture

Clinical Picture

⇒ history of the cause e.g acute pancreatitis.

1. A small pseudo cyst is painless and may be discovered by follow up U/S.
2. A large cyst causes discomfort and manifests as an upper abdominal swelling

Investigations

- Barium meal (lat. View): shows forward displacement of the stomach.
- Abdominal U/S. or CT scan(The most accurate).

Treatment

- Most of cases resolve spontaneously.
- Surgical TTT:
 - indicated if: Persistent cyst $\geq$ 6 wks, cyst $\geq$ 6cm or infected cyst.
 - Methods: The cyst is internally drained by cyst gastrostomy or cystojejunostomy.

Carcinoma of the Pancreas**Incidence**

- Between 55 – 70 years of age.

Etiology

- Exact etiology is unknown.

Pathology

1. **Site :** -cancer head 60% : 2/3 of cases in head proper and 1/3 of cases in periampullary region (ampulla of vatar, duodenal papilla& lower end of CBD).
-cancer body & tail 40%.

2. **Origin:** from acinar & duct cells.

3. **Macroscopic Picture:** Mass infiltrating edge with areas of hge and necrosis.

4. **Microscopic Picture:** Poorly differentiated adenocarcinoma.

Spread

1. **Direct:** to duodenum, CBD, stomach, transverse colon, peritoneum, aorta, IVC.
2. **Lymphatic:** to
 - Celiac LNs.
 - Superior mesenteric LNs.
3. **Blood Spread:** to Liver (mainly), lung, bone&brain.
4. **Transcelomic:** peritoneal nodules, Krukenburg & malignant ascites.

Clinical Picture**A. Cancer Head:**

- 1) Symptoms of Obstructive Jaundice (see later).
- 2) Distended palpable GB in 50% $\square$ (Courvoisier's law).
- 3) Enlarged liver.
- 4) Asthenia (common).

B. Cancer Body or Tail:

➤ *Usually presents late with non-specific symptoms:*

1. **Epigastric pain radiating to the back.**
2. **Occult Manifestations:**
 - Thrombophlebitis migrans (**Trousseau's sign** $\square$).
 - Enlarged left supraclavicular LN. (**Troisier's sign** $\square$).

Investigations

1. For diagnosis:

1. LFTs, stool, urine: see OJ
2. ERCP(biopsy, stent)
3. Barium meal(obsolete): widening of the C-curve of the duodenum
4. Tumor Markers:
 - CA 19.9: recent tumor marker if ↑ indicate irresectable tumor.
 - Carcinoembryonic antigen (CEA).
 - Pancreatic oncofetal antigen (POFA).
 - Pancreatic cancer associated antigen (PCAA).

2. For staging: Abdominal U/S, CTscan, MRI, CXR.....

3. For follow up: tumour markers.

4. For preoperative preparation: CXR, CBC

Treatment

A. Operable cases(less than 10%):

- A. Pre-operative preparation (see O.J.).
- B. Operation:
 - Tumor in head→ Whipple's operation.
 - Tumor in body and tail→ distal pancreatectomy with splenectomy

B. Inoperable cases(more than 90%):→ palliative TTT.

1. Surgical: to relieve the O.J.
 - Endoscopic Stenting(by ERCP or PTC) of the occluded CBD.
 - Triple anastomosis:
 - cholecystojejunostomy:to bypass the occluded CBD.
 - gastrojejunostomy:to avoid further obstruction of the gastric outlet by the tumour.
 - jejuno jejunostomy.
2. Pain relief.
3. Chemotherapy (of low value).

Prognosis

- Extremely poor → 5 years survival rate is < 5 %.

APPENDIX

Acute Appendicitis

Incidence

- It is the **COMMONEST** cause of acute abdomen in young adults
- **Age:** 20 - 30 years of age (If in old age → suspect cancer caecum).
- **Sex:** ♂ : ♀ = 3 : 2

Etiology

Causative Organism:

- E.coli (85%), staph, Strept. Fecalis.

Route of Infection:

- Usually direct from the lumen, rarely blood or lymph.

Predisposing Factors:

- Obstruction ($\frac{2}{3}$ of the cases) by:
 - **Lumen:** hard feces, oxyuriasis, ascaris, FB.
 - **Wall:** tumors of the appendix or caecum but rare.
 - **Outside:** cancer caecum (*in old age*) & adhesions.
- Anatomical factor: as narrow Lumen or wall rich in lymphoid follicles.

Pathology (2 types)

A. Acute Obstructive Appendicitis ($\frac{2}{3}$ of the cases):

- Catarrhal** → mucocoele of the appendix.
- Suppurative** → pyocoele or empyema of the appendix
- Gangrenous** → (common) occurs at the tip or at the site of obstruction → generalized peritonitis (rapid perforation with no time for omental localization)

B. Acute Non-obstructive Appendicitis ($\frac{1}{3}$ of the cases):

Catarrhal, suppurative and gangrenous (less common).

Complications

I. Local complications:

- Persistent Obstruction → Gangrene and perforation → generalized or localized peritonitis.
- Appendicular mass (Phlegmon):
 - When the inflammatory process is slow (> 48 hours).
 - It is suspected by history of pain 3 days ago and temp. 39°.
- Appendicular Abscess:
 - Perforation of the appendix within an appendicular mass forms an abscess.
- Chronic Appendicitis (Recurrent Subacute appendicitis):
 - Usually on top of acute non-obstructive catarrhal appendicitis.
 - **C/p:** Recurrent attacks of abdominal pain and dyspepsia.

5. Fecal Fistula (Usually iatrogenic):

- After drainage of abscess, appendicectomy in presence of mass or associated Crohn's disease.

II. General Complications (rare): Septicemia, toxemia, bacteremia or pyemia.**Clinical Picture**I- Typical Clinical Picture➡ Symptoms:

A-General: FAHM (fever is of low grade).

B-Local:

1- **Pain** (presenting symptom):

It starts periumbilical and ill defined colicky pain then (Within 6-10 hrs) it becomes sharp & localized to the right iliac fossa:

2- **Anorexia & Nausea:** are usually present

3- **Vomiting:** more severe in obstructive type (but infrequent)..

4- **Constipation:** is common.

➡ Signs:

A-General: Mild fever $< 38^{\circ}\text{C}$ & Tachycardia.

B-Local: (more evident on **McBurney's point**)

1. Inspection: rigidity
2. Palpation: guarding, tenderness and rebound tenderness
3. Auscultation: ↓ intestinal movement.
4. DRE: appendix may be felt in the **pelvic type**.

C-Special Signs

1. **Rovsing's sign:** Pressure on left iliac fossa → pain on right iliac fossa.
2. **Hyperaesthesia of triangle of Sheren:** It disappears after perforation
3. **Obturator sign (Zachary Cope):** Pain on internal rotation of flexed RT thigh in pelvic type.
4. **Psoas sign:** Pain on extension of the right thigh in retrocecal type.

II-A Typical forms:

1. **Long Retrocaecal appendix (74%):** pain is minimal but it may touch the ureter → ureteric colic or touch the psoas muscle → +ve psoas sign.
2. **Pelvic appendix (21%):** pain in pelvis, appendix is felt in DRE,
It might touch the Obturator Internus muscle → +ve obturator sign
or touch the Bladder → dysuria or the Rectum → dysentery & diarrhea.
3. **Paracaecal appendix (2%):** classic presentation.
4. **Postileal appendix (0.5%) [Missed Appendix]:** It touches the ileum producing diarrhea (enteritis) then constipation (paralytic ileus)
5. **Subhepatic appendix:** it simulates acute cholecystitis,
but the patient is young + Hyperaesthesia in the triangle of Sheren.

III-Picture of complications:

1-Appendicular mass: History of appendicitis 2-3days earlier

Then Temp. $>38^{\circ}\text{C}$, more tachycardia & vomiting

+ Mass might be felt in RT iliac fossa (under GA).

2-Appendicular abscess: History of appendicitis 5days earlier+S&S of pus loculus

3-Peritonitis: History suggestive of appendicitis,

Then fever over 38°C + signs of diffuse peritonitis(R,T&RT).

D.D.

from other causes of acute abdomen→see D.D. book

Investigations

(It's mainly a **clinical diagnosis**, so investigations are needed mainly to exclude other causes of acute abdomen)

1. **Leucocytic count:** Moderate leucocytosis (10,000 – 16,000/ul)
2. **Urine analysis:** To exclude urinary stones.
3. **Pregnancy test:** If ectopic pregnancy is suspected.
4. **U/S:** May diagnose appendicitis (dilated lumen, thick walled appendix) in few cases+Exclude Gynecological problems, etc.
5. **Laparoscopy:** both diagnostic & therapeutic esp. for females in the childbearing period.

Treatment

I. **Uncomplicated→Appendectomy** (through Gridiron incision at point of maximum tenderness {mostly at McBurney's point})

2. **Complicated**

1-Appendicular mass: Oschner Sherren's regimen(conservative ttt.) then appendectomy after 3 months.

1. Bed rest in Semisitting position.
 2. Ryle if there is vomiting for 48 hours.
 3. Intravenous fluids&antibiotics (Ampicillin + Metronidazole + Aminoglycosides).
 4. **Monitoring of:**
 - Vital signs.
 - Degree of tenderness
 - Vomiting
 - Size of the mass.
- **Results:**
- 1- Mass↓, fever↓, pain↓ → appendectomy after 3 months.
 - 2- Pain ↑ (throbbing), fever ↑ (hectic) → abscess.
 - 3- Pain↓, fever↓, but mass persists → investigate as cancer caecum.

2-Appendicular abscess: drainage under cover of antibiotic then appendectomy after 3-6 months.

3-generalized peritonitis: R&M, laparotomy, peritoneal toilet, appendectomy and drainage.

PERITONEUM

Acute (2ry) Bacterial Peritonitis

Etiology

1- Causative Organism:

- Gram -ve: E.coli (**COMMONEST**).
- Gram +ve: Staph, Strept, Pneumococci.
- Anaerobe: Bacteroids, Clostridium welchii.

2- Route of Infection:

1- Direct Spread From :

- a- Infected organs: e.g. appendicitis, cholecystitis or diverticulitis
- b- Leaking organs: e.g. perforated PU, leaking anastomosis
- c- Through an operation or a traumatic wound.

2- Blood Spread: may occur in cases of septicemia and pyemia (rare)

Pathology & Fate

1. Resolution if the source of infection is controlled or removed.
2. Localization & abscess formation either around the 1ry viscus or in an anatomical compartment.

➤ Factors which facilitate Localization of Infection:

1. Anatomical:

The peritoneal cavity is divided into 2 compartments(greater&lesser sacs)

2. Peristalsis:

- Reduced & this helps in preventing spread of the infective process.

3. Greater Omentum:

- Becomes adherent to the inflamed structures (policeman of the abdomen).

4. Peritoneal Fluid:

- Rich in PNL and antibodies.

3. Flaring up → diffuse peritonitis.
4. septicemia → death

Diffuse (2ry) Bacterial Peritonitis

Clinical Picture

Symptoms

A-General: FAHM

B-Local: history of the original cause+

1- Pain:

- The site of maximum pain is at the site of the original lesion.

2- Abdominal Distension.

3- Vomiting.

4- Absolute Constipation(no faeces or flatus).

Signs

- 1- General: high temp.& tachycardia.

2- Local:

1. Inspection:
-Limited abdominal movements with respiration all over the abdomen
2. Palpation:
-Guarding, tenderness and rebound tenderness all over the abdomen.
3. Percussion: Shifting dullness.
4. Auscultation:
-Absent or diminished intestinal sounds (dead silent abd).
5. PR / PV: fullness in Douglas pouch in ♀ or rectovesical pouch in ♂.

Causes of death & complications of Generalized Peritonitis

- 1) Toxemia, septicemia, pyemia, bacteremia.
- 2) Multiple organ failure.
- 3) Paralytic ileus.
- 4) H₂O and electrolyte imbalance.
- 5) Wound infection.
- 6) Residual abscess.

Investigations**I. For diagnosis:**

- U/S: Intra-abdominal fluid collection.
- Peritoneal diagnostic aspiration:
 - May be helpful in determining the nature of the fluid e.g.:
 - Bilious → perforated DU.
 - Pus → bacterial peritonitis.
- CBC: leucocytosis with shift to Lt.

2. For the cause:**-Plain X- Ray abdomen:**

May indicate the cause (e.g. air under diaphragm in perforated viscous).

Treatment**I. Resuscitation & Monitoring:****A-****Resuscitation:**

1. Ryle: for continuous suction.
2. IV line: Fluids, blood, antibiotics & analgesics
3. Catheter: to detect urine output.

B-**Monitoring: BP, pulse, urine output, CVP, electrolytes and pH.****2. Exploratory Laparotomy:**

- 1- Incision: Midline incision in doubtful cases.
- 2- Peritoneal toilet.
- 3- Identification of the cause.
- 4- Drainage
 - Hepatorenal drain: most dependent area while lying down.
 - Pelvic drain: most dependent area during standing.
 - Drain beside the cause.
- 5- The abdomen is closed.

Localized 2ry Bacterial Peritonitis

- 1- Subphrenic abscess.
- 2- Iliac abscess.
- 3- Pelvic abscess.

Subphrenic Abscess

It is accumulation of pus under diaphragm.

Etiology

- **Causative Organism:**
 - E. coli, Staph. or Strept.
- **Route of Infection:**
 - A-direct Spread(mainly) :**
 1. Residual pus collection following generalized peritonitis.
 2. Inflamed or perforated viscera e.g. appendix, GB.
 3. Postoperative collection after appendectomy or cholecystectomy.
 - B-Lymphatic spread:** from chest infection.

Clinical Picture

Symptoms

- **General:**
 - FAHM.
- **Local:**
 - 1- Persistent **hiccough** [□].
 - 2- **Pain** is slight or absent
 - **Site:** in the epigastrium or in the Rt. Hypochondrium.
 - **Referred to:** the shoulder. -Increases by: inspiration.

Signs

- **General:**
 - Hectic fever, tachycardia, may be signs of pleural effusion.
- **Local:**
 1. **Inspection:** rigidity over the site of abscess.
 2. **Palpation:** -Gaurding, T&RT in the lower ribs & just below the costal margin.
 - Downward displacement of liver
 3. **Percussion:**
 - If the abscess contains gas, **4 percussion zones** may be elicited:
 - a-Resonance of the lung.
 - b-Dullness of the pleural effusion.
 - c-Resonance of the gas in the abscess.
 - d-Dullness of the liver or pus in the abscess.
 4. **Auscultation:** Diminished intestinal sounds

Investigations

- **Abdominal U/S & CT:** The best localizing method.
- **Chest X-Ray:**
 - Tented diaphragm, pleural effusion, may be air under diaphragm.
- **CBC:** ↑ TLC & shift to the left.

Treatment

1. **Conservative** (*early*): Rest, Analgesics, Antibiotics, Antipyretics.
2. **Percutaneous Drainage** (U/S or CT guided):
 - In case of:
 1. Failure of conservative treatment.
 2. Evidence of suppuration.
3. **Open Drainage: extrapleural extraperitoneal approach.**
 - **Indication:**
 - Abscess with multiple loculi or thick pus or with rapid reaccumulation.

TB Peritonitis**Etiology**

- It is always secondary to a tuberculous focus elsewhere.

Route

1. **Direct:** (intestinal TB - tabes mesenterica – TB salpingitis) COMMONEST.
2. **Hematogenous:** e.g. pulmonary TB.
3. **Lymphatic:** e.g. from pleura or bowel.

Pathology

1. **Ascitic:** *commonest*
 - Tubercles + fluid (+) lymphocytes & tubercle bacilli.
2. **Encysted:** *localized form of ascitic type*
3. **Adhesive:**
 - Omentum is rolled-up (Mass).
 - Adhesive Intestinal obstruction.
4. **Purulent (Caseous):**
 - Rare → in young females (as a complication of TB salpingitis).
5. **Acute Miliary type:**
 - It resembles acute peritonitis and the diagnosis is frequently made during operation.

Clinical Picture

► More common in children and young adults

- **Symptoms of TB toxemia:**
 - Night fever, night sweat, loss of weight & loss of appetite.
- **Recurrent attacks of non-specific abdominal symptoms:**
 - Pain (vague), colics, diarrhea, distension due to toxemia, TB enteritis.
- **Abdominal examination shows:**
 - **Palpation:**
 - Doughy abdomen, generalized tenderness with mild rigidity, palpable mass: rolled omentum "sausage-shaped", TB LNs.
 - **Percussion:**
 - Free ascites (usually small volume).
- **PV:** may show tubo-ovarian mass.

Investigations

► The most important diagnostic tools are:

- CBC.

1. Laboratory:

1. CBC: anemia, lymphocytosis and high ESR.
2. Tuberculin test: +ve.

2. Radiological:

1. Plain CXR: may reveal a primary focus.
2. Abdominal U/S: ascites free or encysted, LNs, no liver or spleen affected.

3. Instrumental:

1. Abdominal Tapping:
2. Laparoscopic: most reliable method of diagnosis of TB peritonitis.
3. Exploratory laparotomy.

Treatment *Mainly medical*

- At least 2 antituberculous drugs for at least one year.
- Surgery is indicated in few cases such as (Intestinal obstruction – doubtful diagnosis – cold abscess).

SMALL INTESTINE

Meckel's Diverticulum

Definition

- Persistent patency of the proximal part of the vitellointestinal duct.

Incidence (Rule of 2)

- 2% of people are affected.
- 2% have complications.
- 2 feet from ileocecal valve.
- 2 inches long.

Pathology

- It is a true diverticulum has a separate blood supply from SMA.
- It may contain ectopic tissues.

Clinical Picture

- Asymptomatic.
- The most frequent manifestation is bleeding.

Complications

1. Intestinal Obstruction (COMMONEST):
It may be due to: **Intussusception or volvulus.**
2. Peptic ulceration:
 - Sometimes the diverticulum contains ectopic gastric epithelium.
 - This leads to formation of peptic ulcer on the neighboring epithelium.
3. Meckel's Diverticulitis:
 - **Diagnosis**:
 - Clinical picture similar to that of acute appendicitis.
 - At operation, the appendix is found normal (it is the only way to differentiate between acute appendicitis and Meckel's diverticulitis).
4. Littre's Hernia
 - May strangulate causing strangulation without obstruction.

Investigations

- Barium meals follow through: may show a diverticulum.
- Tc⁹⁹ Scan + gamma camera: safer than arteriography can demonstrate gastric mucosa.

Treatment

- **Symptomatic cases**:
 - Wedge resection of the ileum and closure of the defect transversely or segmental resection is indicated.
- **Silent cases and accidentally discovered at laparotomy**:
 - Indications of resection (if complications are likely):
 - 1- Narrow mouthed.
 - 2- The wall of the diverticulum is thickened.
 - 3- Attached band of adhesions.
 - 4- Young adults and children.

Small intestinal fistula

Etiology

- Injury during operation (in majority of cases)
- Crohn's disease.
- Colonic diverticulitis.
- Cancer.
- Radiation enteritis.
- Intestinal tuberculosis.
- Mesenteric vascular disease.

Types

- 1- Internal fistula : connects intestine to another hollow viscus
- 2- External fistula:
 - **High (>500 ml / day)** and **low (<500 ml / day)** fistulas depending on their location and output.
 - The more proximal the fistula the more the output, the more serious the problem.

Complications

- Sepsis (intraperitoneal abscess).
- Skin necrosis.
- Malnutrition and fluid and electrolyte abnormalities.

Investigations

- Barium meal follow through.
- Barium enema.
- Fistulogram.

Treatment

Conservative management

- Nutritional support by I.V fluids
- Control of sepsis
 - Sumb suction helps to control sepsis and provide drainage for associated intra-abdominal abscess.
 - Nasogastric tube in patient with ileus helps to reduce output from fistula.
 - Somatostatin also inhibits intestinal secretion and motility.
 - Systemic antibiotics should be given until sepsis is controlled.
- Skin stoma care
- With modern conservative management, spontaneous closure occurs in 70-80% of cases and mortality averages 6-10%.

Surgical treatment

A. External fistula :

➤ indications :

1. If there is still significant output after 4-6 weeks of conservative management,
2. Distal obstruction.
3. Affected segment has active granulomatous disease.

B. Internal fistula:

- Spontaneous closure is rare
- Surgery for:
 - 1- fistula with urinary tract
 - 2- if the fistula passes a long segment

COLON & RECTUM

Congenital Megacolon (Aganglionic Megacolon) (Hirschsprung Disease)

Etiology

- Unknown.

Pathology

- The disease starts in the upper part of the anal canal and extends proximally to the recto sigmoid area in 80% of cases (typical cases) or it may involve variable lengths of the colon or even the whole colon.

Spastic segment	Transitional one	Proximal dilated one
Absent ganglia	Few ganglion cells	Normal ganglion cells

- There are 3 segments:
- Microscopic:
 - Absence of ganglia of Auer Bach's and Meissner's plexus.

Clinical Picture

Any new born presenting with delayed passage of meconium for more than 24 hrs should be considered as having Hirschsprung's Disease until proved otherwise

➤ The most common presentation:

Symptoms

Chronic constipation:

- Delayed passage of meconium > 24 hrs.

Signs

- Inspection:
 - Abdominal distension, visible peristalsis.
- DRE:
 - Empty rectum
 - Grips on the finger.
 - Characteristic gush of foetid stool on withdrawal of the finger[☑].

Complications

➤ Of severe cases :

General:

- Delayed growth and development.
- Chest infection.

Local:

- Obstructive toxic enterocolitis (fever, diarrhea and abdominal distension)
 - **cause of death.**
- Acute obstruction.

➤ Of moderate cases : failure to thrive

Investigations

1. Imaging:

▪ Barium Enema:

• Finding:

- Will show narrow aganglionic segment with marked proximal colon dilatation.
- Its accuracy is 80%.

➤ The most important diagnostic tools are[☑]:

- Barium enema

2. Instrumental:

▪ Rectal biopsy: (most diagnostic[☑])

- Shows absence of ganglion cells.

▪ Anorectal manometry:

- Reveals failure of relaxation of anal sphincters in response to rectal distension.

Treatment

Patients presenting by obstructive enterocolitis

Conservative Treatment

- 1- NGT suction.
- 2- IV fluids.
- 3- Repeated colon wash-outs with saline.

Obstruction is relieved

↓
Prepare for later elective surgery

Obstruction is not relieved

↓
Urgent colostomy

Patients without intestinal obstruction

Elective surgery after preparation

Preparation:

- 1- Repeated enemas.
- 2- Prophylactic antibiotics
- 3- Correction of dehydration.

Definitive Surgery:

Timing: ideally at 6-9 months, 10 Kg, Hb 10 gm/dl

Procedure:

- Swenson's operation

Diverticular Disease of the Colon

Incidence

- Common in western countries[□] (50% above 65 years).

Etiology

- Chronic constipation due to low fiber diet.

Pathogenesis

- Chronic constipation → ↑ intraluminal colonic pressure → muscle spasm and incoordination → herniation of colonic mucosa (pulsion diverticulae).

Pathology

Site

- Sigmoid[□] colon in 90% of cases.
- Any area of the colon may be involved.
- Rectum is never affected.

Clinical Picture

- Asymptomatic 80% (stage of diverticulosis).
- I- Complicated cases: (10-25%)
 - A- Acute diverticulitis:
 - As acute appendicitis but on the left side.
 - B- Chronic diverticulitis:
 - Long history of recurrent attacks of abdominal pain with passage of blood and mucous per rectum.
 - C- Bleeding per rectum:
 - Massive bright red bleeding

Complications

- Acute diverticulitis: (Try to obstruction of its neck) and its complications.
 - Perforation.
 - Fistula formation: vesico-colic or vagino-colic.
- Chronic diverticulitis: → colon stricture and adhesive intestinal obstruction.
- Bleeding per rectum.

DD

- Cancer colon.
- Angiodysplasia of the colon.

Investigations

I- Radiological:

- Barium enema:
 - Early: Saw-tooth appearance
 - Late: Well-developed diverticula can be visualized.
- CT scan:
 - Best investigation in acute diverticulitis.

2- Instrumental:

- Sigmoidoscopy:
 - Will detect the mouths of diverticulae.

The most important diagnostic tool is:

- Barium enema

Treatment

1- Treatment of uncomplicated cases:

1. High-fiber diet.
2. Anti-spasmodics for the abdominal colic.

2- Treatment of complicated cases:

A- Acute diverticulitis:

- Hospital admission, bowel rest, antibiotics.

1. If complicated diverticular abscess:

- Incision and drainage in huge multilocular abscess.
- Pericolic abscess: percutaneous U/S guided drainage, then resection in patients who remain symptomatic or who develop recurrent abscess.

2. Generalized peritonitis:

- Resuscitation and urgent laparotomy.
- Peritoneal toilet and drainage.
- Resect the perforated colon by a Hartmann's procedure.

3. colo-vesical Fistula:

- Resection with repair of the fistula.

B- Chronic diverticulitis.

- Colectomy after proper preparation of the colon.

C- Bleeding per rectum:

- Resuscitation followed by angiography or Tc99 to localize the site of bleeding:
- If localized → regional resection
- If localization fails → total colectomy + ileorectal anastomosis.

Rectal Prolapse

Definition

- Abnormal descent of part or all of the upper rectum through the lower rectum and/or the anal canal to protrude outside the anus.

	Partial RP	Complete RP
Definition	Prolapse of rectal mucosa	Prolapse of whole thickness of the rectal wall
Etiology	<u>In children</u> 1. Loss of curve of sacrum, so the rectum and anus form a vertical tube. 2. straining at defecation. <u>In adults</u> 1) advanced cases of piles. 2) Sphincteric atony in elderly	▪ More common in females, while in Egypt more in young males (due to bilharzial colitis). 1. C.T. disease (d.t. defective collagen synthesis). 2. Abnormal mobility of the mesorectum . 3. In Egypt, Bilharzial proctitis and colitis → continuous tenesmus → strangulation
Pathology		
Length	< 5 cm	> 5 cm
Thickness 	Mucosa only	Whole rectal thickness

	Partial RP	Complete RP
Complications	1- Ulceration and infection. 3- Irreducibility. 5- Fecal incontinence.	2- Bleeding. 4- Strangulation and gangrene. 6- Discharge, pruritus.
Clinical Picture	Something protruding from the anus at defecation.	
Investigation	➤ Anorectal manometer, sigmoidoscopy.	
Treatment	Children 1. Correction of the cause 2. Digital reposition → if failed ↓ 3. Submucous injection of absolute alcohol or phenol in almond oil → induces fibrosis 4. Thiersch operation (perianal circlage) Banding or excision of redundant mucosa.	Adults: 1. Correction of the cause 2. sphincter exercises 3. Excision of the prolapsed mucosa There are various surgical procedure include: <u>1- Rectopexy:(laparoscopic)</u> The rectum is mobilized and pulled up, then fixed to a mesh attached to pre-sacral (Waldeyr's) fascia and puborectalis muscle by sutures. <u>2- Excision</u> of the redant rectum . <u>3-Delorme's operation.</u> <u>4. Thiersch operation</u> <u>5. Perineal rectosigmoidectomy.</u>

Familial Polyposis Coli (FPC)

Etiology

- Autosomal dominant or may be part of Gardner's syndrome.

Pathology

Site

- The sigmoid colon and rectum[□] are the commonest sites.
- They are full of multiple polyps (at least 100).

Macroscopic

- The polyps are sessile and pedunculated.

Microscopic

- Three histological types are recognized; tubular, tubulovillous and villous.

Clinical picture

- Bleeding and mucous per rectum and diarrhea.
- PR will reveal the polyps.

Investigations

- Barium enema: multiple filling defects.
- Lower GI endoscopy and biopsy.

Treatment

- Total prctocolectomy and ileal reservoir.

Carcinoma of the Colon and Rectum

Incidence (epidemiology)

- **Age:** - Usually above 50 years but not rare before this age.
- **Sex:** - Cancer cecum more common in females
- Otherwise → more common in males

Etiology & predisposing factors

A. CHRONIC IRRITATION:

1. Dietary factors: low fiber and high animal protein
2. Smoking, alcohol.
3. Uretero-colic anastomosis.
4. IBD (ulcerative colitis) → if pan-colitis > 10 years.

B. BENIGN PRECANCEROUS LESIONS:

- Colonic polyps especially villous adenoma more than 2 cm.

C. GENETIC FACTORS:

FPC and Gardner syndrome → 100% precancerous by the 5th decade, more on Lt. side.

Pathology

Site

- 2/3 of cases are situated in the rectum and sigmoid colon[□].
- 25 % in the caecum and the right colon (10% in cecum).
- **Multiple tumors in 5% of cases.**

Macroscopic Picture

- Annular infiltration, ulcerative or fungating mass.

Microscopic Picture

- Adenocarcinoma, colloid or anaplastic varcinoma.

Spread

1- Direct spread:

First in the colonic wall itself, then to the neighboring organs.

2- Lymphatic spread:

- ➔ In the right and transverse colon: into epicolic, paracolic and then into superior mesenteric L.Ns.
- ➔ In left colon: into epicolic, paracolic and then into inferior mesenteric L.Ns
- ➔ Nodal involvement is found in 40% of case coming to operation.

3- Blood spread:

- ➔ mainly to the liver (hepatic metastases is found in 20% of cases coming to surgery).

4- Transperitoneal spread.

Complications

- 1- IO occurs in 20 % of cases esp . with left colon tumors.
- 2- Perforation or the formation of enterocolic or vesico-colic fistula.
- 3- Fistula formation (malignant fistula), either with bladder or vagina.
- 4- Bleeding, chronic bleeding is the rule while massive bleeding is rare.
- 5- Complications due to spread e.g: ascites, jaundice.....

Clinical Features

A. WITHOUT OBSTRUCTION

✚ Lt. colon cancer

1. Change of bowel habits (it is the most important presentation): usually progressive constipation, constipation alternating with diarrhea or spurious diarrhea.
2. Bleeding per rectum: fresh, but not massive.
3. Symptoms of intestinal obstruction (especially with sigmoid cancer): either acute, subacute or chronic.
4. Mass on the Lt. side of abdomen.

✚ Rectum and sigmoid colon

1. Bleeding per rectum, tenesmus.
2. DRE → palpable if within 10 cm from the anal verge.

✚ Transverse colon

- Dyspepsia, anemia, epigastric mass.

✚ Caecum and ascending colon

Unexplained loss of weight & appetite, mass and anemia.

NB: Whenever rectal bleeding occurs in a middle aged or older individual even in the presence of piles → co-existing rectal cancer should be excluded.

B. WITH OBSTRUCTION

- ✚ Commonest with left sided cancers, rare with other types.

Investigations

1. Radiological

1. **BARIUM ENEMA** (better a double contrast barium enema):
 - The cauliflower tumor appears as a fixed irregular filling defect.
 - Annular strictures of the left colon show a characteristic "apple core appearance" [□].

2. SPIRAL CT AND U/S.

2. Instrumental

3. **ENDOSCOPY** (sigmoidoscopy if < 60cm from anus, colonoscopy if more) reaching up to caecum:
 - Mandatory in every patient above 40 years with complaint of piles.

3. Laboratory

- CBC, CEA.

Treatment

A. Without obstruction

I. Operable cases (curable):

1. Preoperative preparation:

2. Elective radical resection:

- Operations (according to the site):

a) Tumors of the caecum:

- Rt. hemicolectomy

b) Tumors of the ascending colon or hepatic flexure:

- Extended Rt. hemicolectomy.

c) Tumors of the transverse colon:

- Transverse colectomy.

d) Tumors of the splenic flexure or descending colon:

- Lt. hemicolectomy.

e) Tumors of the sigmoid colon:

- Sigmoid colectomy.

f) Tumors of the rectum:**1- Tumors of the upper third of the rectum:**

Treated by anterior restorative resection.

2- Tumors of the lower third of the rectum:

Treated by an abdominoperineal resection (of Miles).

3- Tumors of the middle third of the rectum:

Used to be treated by abdominoperineal resection and a colostomy like lower tumors. Nowadays it is treated by anterior restorative resection.

g) Liver metastasis:

- Resection if <4 , limited to one lobe and no other metastasis.

II. Inoperable cases:

- Resectable: resection is preferred as it is adenocarcinoma (insensitive to radiotherapy or chemotherapy)
- Irresectable: operation is done to avoid obstruction
 - Right colon → side-to-side ileo-transverse anastomosis.
 - Left colon and rectum → proximal colostomy.

B. With obstruction:

- If good general condition → exploration
 - Operable → radical resection according to site.
 - Inoperable → surgery to avoid obstruction later.
- If bad general condition (unfit for surgery) → colostomy → improve general condition → colon resection.

Prognosis**Worse prognosis of the colorectal cancer is encountered in the following conditions:**

1. Young age at clinical presentation.
2. Colonic obstruction or perforation.
3. Blood vessel or lymphatic obstruction.
4. Liver and/or distant metastases.
5. Aneuploidy.

ANAL CANAL

Imperforate Anus

Types

1. High anomalies (More common)

- Failure of the rectum to pass through the pelvic floor.
- Associated with fistulous communication with posterior urethra in males or vagina in ♀.

▪ Types:

a- Anorectal Agenesis:

- Rectum: Forms a blind pouch above the pelvic floor.
- Anal canal: absent

b- Rectal Atresia:

- Rectum: Forms a blind pouch above the pelvic floor
- Anal canal: Forms a blind pouch below the pelvic floor

2. Low anomalies (Less common)

- Distal to the pelvic floor.
- No defect in anal sphincter
- a- Covered Anus:
 - The anal canal is covered by skin bar and usually opens into an ectopic site anterior to normal position.
- b- Membranous Anus:
 - There is a membrane at the dentate line → retained meconium → bulging of this membrane.

Associated anomalies

- **VACTERL**, more common in high than low anomalies.

Clinical Picture

General examination

- To exclude associated congenital anomalies.

Abdominal examination

- There may be evidence of intestinal obstruction.

Examination of the perineum

1. Presence of anus, its size and site
2. Presence of an anal dimple
3. **If there is an impulse on crying at the site of the anus → low anomaly.**
4. Subcutaneous fistulous track full of meconium → ectopic anus
5. **Meconium at the tip of the penis → fistula to the bladder or urethra.**
6. If the thermometer can be introduced into the anus for > 1 cm, this excludes rectal atresia.

Investigations

I. FOR DIAGNOSIS: Radiological:

▪ Plain X-ray (invertogram):

- 24 hours after birth
- We can differentiate between high and low anomalies by:
 - A. Measuring the distance between coin and distal gas-shadow:
 - ⇒ If < 1 cm → low anomaly.
 - ⇒ If > 1 cm → high anomaly.

▪ IVP: (In high anomalies)

Treatment**1. High anomalies:**⇒ **Treated with staged surgery:**

- First stage: Temporary colostomy
- Second stage: Anorectal pull-through (abdominal approach)
- Third stage: closure of colostomy.

2. Low anomalies:

- Only perineal surgery

3. Treatment of associated anomalies if found

Pilonidal Sinus

Definition

- Subcutaneous granulation cavity described as a nest (latin-nidus) containing hair (latin-pilus) and connected to the skin by midline openings (latin-sinus).

Incidence

- More in young adult males with dark dense strong hair

Etiology➤ The actual cause is unknown[□], possible theories are:**A. Congenital theory**

- Infection of congenital dermal appendages overlying the lower sacral region that presents after puberty[□].

B. Acquired theories

- Jeep bottom theory[□]: prolonged sitting on hard seats changes the direction of the hairs.
- Loose hairs from head or back fall on the skin over the sacrum and coccyx, as a result of pressure from sitting, rolling movement of the buttocks, sweat and poor hygiene; the hairs are drawn through the skin to accumulate in subcutaneous tissue.

Pathology➤ **Sites:**

- 1-Upper part of anal cleft (most common[□]).
- 2- Others: Axilla, umbilicus, Interdigital (in barbers), suprasternal notch.

Clinical Picture**Symptoms**

- 1- May be asymptomatic.
- 2- Local discomfort, **discharge**[□] (the most important, characteristically foul containing hair).
- 3- Painful swelling if abscess is formed.

Signs

1. Discharging midline and lateral openings with loose hairs protruding from it.
2. If abscess is formed: redness, tenderness and pus oozing from the openings.

Treatment**A. Pilonidal Abscess**

- Rest, antibiotics, antipyretics, anti-inflammatory.
- Abscess is drained → Removal of loose hairs.
- The wound is left open to heal with granulation tissue.

B. Pilonidal Sinus

- Sinuses are identified by methylene blue or hydrogen peroxide.
- Different options are available:
 1. **Laying out of the cavity and side tracks:** The resulting wound is left open, packed and is allowed to heal by 2ry intention.
 2. **Localized excision of the cavity and side tracks**
 3. **Wide excision of the skin and subcutaneous tissue** down to periosteum of the sacrum should no longer be practiced.
 4. **Another option: (Conservative surgery)**
 - Laying out the cavity and side tracks and curettage:
 - The resulting wound is left open, packed and is allowed to heal by secondary intention.

Anal Fissure

Definition

- An **elongated ulcer** which occurs in the **long axis** of the **lower anal canal**.

Etiology

- A. No definitive cause: is found in the **majority**, postulated mechanisms are:
 - Trauma: by hard stool or repeated deliveries.
 - Ischemia:
- B. Definitive cause: is found in a **minority**.
 - IBD especially Crohn's disease, STDs
 - Iatrogenic: large enema, post-hemorrhoidectomy.

Pathogenesis

- *Pain is the main pathogenic factor in acute fissure.*

Pathology**Site:**

- **Usually in the midline.**
 - Midline posteriorly (90%)[□]
 - Midline anteriorly (10%): Especially common in multiparous females.
- **May be anywhere** as in Crohn's disease.

Clinical Picture**Symptoms**

1. Pain[□]: sharp, starts at defecation and ends suddenly after 1 hour.
2. Constipation.
3. Bleeding: slight bright red on the surface of the stool.
4. Slight anal discharge and pruritis.
5. Reflex symptoms: Burning micturition, dysmenorrhea and pain along the thighs

Signs➤ In acute anal fissure:

- Inspection: The anal verge is tightly contracted, puckered anus
- DRE: Better to be avoided[□], as it is very painful

➤ In chronic fissure:

- Inspection: An anal papilla or a sentinel pile may be present
- DRE: Fissure is fibrotic & indurated (*Button hole induration*)

DD**Of painful anal conditions**

1. Anal fissure.
2. Perianal suppuration.
3. Prolapsed strangulated piles.
4. Acute perianal hematoma.
5. Carcinoma of the anus.
6. Proctalgia fugax (idiopathic).
7. Crohn's disease

Complications

- 1- Fissure abscess → fistula.
- 2- Acquired megacolon.

Investigations

- It is a clinical diagnosis, however, if multiple and in uncommon sites
→ Search for a specific cause e.g. Crohn's disease → biopsy

Treatment**I. Acute Fissure**

- **Essentially medical but sometimes surgery is needed**
- ❖ **Life style changes:**
 - High fiber diet, laxatives, sitting in a warm bath
 - ❖ **Medical treatment:**
 - Chemical sphincterotomy by:
 - ⇒ Glyceryl trinitrate (GTN) ointment (2-10%):
 - ⇒ alternatives:
 - Ca channel blockers (diltiazem, nifedipine)
 - local injection of botulinum toxins
 - A local anesthetic ointment is introduced gently as 2 % or 5% lignocaine.
 - Local steroids (hydrocortisone) to relieve pain & inflammation.
 - ❖ **Surgical treatment:**
 - **Lateral sphincterotomy**[□]:
 - Indicated if all previous measures fail to relieve pain within a few days.

II. Chronic Fissure

- ❖ **Treatment is surgical**
 - If the fissure is not so old, a lateral internal sphincterotomy operation is very successful.
 - If the fissure is very fibrosed with a sentinel pile, the best procedure is to do fissurectomy and posterior internal sphincterotomy.

Hemorrhoids (Piles)

Types

- A- Internal piles = above dentate line and covered by mucosa.
- B- External piles = below dentate lined and covered by skin.
- C- Interno-external piles (the most common).

Internal piles

Definition

- Dilated tortuous superior hemorrhoidal plexus of veins.

Etiology

- Primary hemorrhoids (most common)

Predisposing factors: (congenital weak mesenchyme)

I- Morphological factors:

- Weight of blood column unassisted by valves → high venous pressure in the rectum.

II- Anatomical factors: superior rectal vein radicals.

- a- Lie unsupported in the loose submucous connective tissue → may prolapse
- b- Have no valves → high venous pressure

Exacerbating factors

A. Primary hemorrhoids

- Straining (e.g. with constipation): very potent cause
- Diarrhea & dysentery: less important cause.

B. Secondary hemorrhoids

- Pregnancy, cancer rectum or portal HTN (anorectal varices).

Pathology

Types & sites

- **Mother piles:** Present at 3, 7, 11 o'clock in lithotomy position.
- **Daughter piles:** Present in between mother piles.

Clinical Picture

Symptoms

1. Bleeding per rectum:

- **The most common[□] and earliest complaint.**
- Bleeding is characterized by: painless and occurs at the end of micturition.

2. Prolapse.

3. Pruritus and anal discharge.

4. Pain: only in complicated cases[□].

Signs

1. Inspection:

- Prolapsed piles (4th degree).
- Unprolapsed piles may come into view with straining.

2. DRE:

- Internal piles cannot be felt (compressible) unless thrombosed.
- It is essential to exclude cancer rectum.

➤ Clinical Degrees of Piles According to Prolapse

1. First degree

- The patient has **only bleeding** but no prolapse of piles, diagnosed only by **proctoscope** as it is not felt by DRE. It is not reaching level of dentate line.

2. Second degree

- The piles prolapse only during defecation, but they are spontaneously reduced at the end of act.

3. Third degree

- There is prolapse of piles during defecation and patient has to reduce it manually.

4. Fourth degree

- There is permanent prolapse of piles.

Complications

1. Bleeding per rectum.
2. Thrombosis (thrombophlebitis of strangulated piles if not reduced within 1-2 hours)
3. Fibrosis
4. Ulceration of the mucous membrane.
5. Gangrene: if strangulation occludes arterial supply.
6. Suppuration: if secondary infection occurs → abscess, bacteremia.
7. Pyemia: (portal)
8. Partial rectal prolapse (*piles are the most common cause*)
9. strangulation.

Treatment

Primary Hemorrhoids

➤ For first and second degrees:

- Conservative TTT:
 1. High fiber diet
 2. Small doses of laxative.
 3. Suppositories that contain decongestant.
- Injection sclerotherapy☑:
- Injection of irritant material (e.g. 5% phenol in almond oil) in the submucosa to induce fibrosis, obliteration of venous plexus.
- Rubber band ligation (good for large 2nd degree piles☑):
 - Placing a rubber band around the piles → ischemic necrosis & separation.
- Photocoagulation:
 - Raise the tissue temperature to 100°C → coagulative necrosis & separation.
- Cryosurgery (not done now):
 - Using liquid nitrogen (-196°C) → coagulative necrosis of piles & separation

➤ For third and fourth degrees:

- Hemorrhoidectomy:.

Secondary hemorrhoids

- Treatment of the cause.

Complicated piles

➤ Strangulated piles:

- If the case is diagnosed early: surgical intervention under antibiotic.
- If the case diagnosed late: rest in bed, antibiotics, analgesics, decongestive ointments and local compresses by lead subacetate lotion.

Acute External Piles (peri-anal hematoma)

Pathogenesis

- Rupture of a dilated anal vein secondary to straining at defecation, coughing or lifting a heavy weight → hemorrhage in the loose subcutaneous connective tissue.

Clinical Picture

Symptoms

- Sudden severe pain

Signs

- Tense, tender, bluish swelling covered by smooth shining skin

Fate

- Untreated hematoma usually resolves
- It may suppurate, ulcerate through skin or fibrose and give rise to a cutaneous tag.

Treatment

- Under local anesthesia, the hemorrhoid is bisected and the 2 halves are excised with small position of adjacent skin to leave a pear shaped wound, which is left open to granulate.

Anal Fistula

Definition

- It is a granulomatous track which opens:
 - Internally → into the rectum or the anal canal, usually at the level of dentate line.
 - And
 - Externally → on the skin around the anus.

Etiology

- Infection of anal glands → abscess formation followed by:
 - Spontaneous rupture or
 - Inadequate drainage

Classifications

A. Standard classification:

- According to position of internal opening in relation to anorectal ring:
 1. Low fistula: below the anorectal ring.
 2. High fistula: above the anorectal ring.

B. New classification (Park's classification)

- According to the course of fistulous track:
 - A- An intersphincteric fistula tracks (most common 70%).
 - B- A transsphincteric fistula.
 - C- A suprasphincteric fistula .
 - D- An extrasphincteric fistula.

C. Goodsall's rule[□]:

- 1- If external opening is posterior to transverse anal line:
 - The fistulous track is curved (hoarse-shoe)
 - There is a common internal opening in *midline posterior*.
- 2- If external opening is anterior to transverse anal line:
 - The fistulous track is **straight**.
 - The internal opening is **separate** on the same.

Clinical Picture

Symptoms

- History of a previous perianal abscess.
- Followed by **purulent discharge** (the most important).
- Local sourness and pruritis ani.
- Perianal pain & swelling if abscess develops.

Signs

- **Inspection:**
 - External opening may be seen as small skin elevation surrounded by granulation tissue.
- **Probe examination should be delayed** and done preoperatively under anesthesia as it is painful and causes reactivation of infection and formation of new tracks.

Investigations

A. Fistulogram: (rarely done)

- With lipidol → demonstrate track & any side branches

B. Proctoscopy:

- Internal opening may be seen

C. Recently: endoluminal U/S or MRI.

Treatment

1. Fistulotomy for an Intersphincteric fistula:

- Division of part of the internal sphincter for deroofting of the fistulous track (no disturbance of continence mechanism).

2. Fistulotomy for Transsphincteric fistula:

- Division of part of the internal sphincter and superficial part of the external sphincter for deroofting the fistulous track (minor disturbance of continence mechanism)

3. Fistulotomy for suprasphincteric fistula:

➤ 2 stage operation:

- First stage:
 - Deroofing the lower part of intersphincteric component of the fistulous track by dividing the internal sphincter.
- Second stage:
 - The rest of the track is laid open guided by Seton suture which may be left in place to induce fibrosis then open it.

4. Extrasphincteric fistulae:

- Proximal colostomy and staged Fistulotomy then treat the underlying cause.

➤ Fistulectomy:

- Is complete excision of the fistulous track.

Anorectal Abscess

Organism

- E. coli.

Etiology

- ⇒ Bad general condition.
- ⇒ Bad hygiene.
- ⇒ Persistence of predisposing factors.

➤ Primary anorectal abscess: Is due to:

1. Infection of the anal glands☐:

➤ Secondary anorectal abscess:

1. Inflammatory bowel disease.
2. Specific infections e.g. TB.
3. Anorectal carcinoma.
4. Infection of a perianal hematoma, thrombosed piles or anal fissure.

Classification and etiology:

	Perianal (subcutaneous) abscess 60 %☐	Ischiorectal abscess 30%	Submucous abscess 5%	Pelvirectal abscess 5%
Site	Subcutaneous close to the anus	Ischiorectal fossa	Submucosa above dentate line	Between the upper surface of levator ani and pelvic peritoneum

Clinical Picture

Symptoms

- General: fever, headache, anorexia and malaise
- Local:
 - Continuous, throbbing pain, aggravated by defecation.
 - Painful swelling present only on perianal type.

Signs

- Local
 - 1- Perianal: red, hot and tender swelling.
 - 2- Ischiorectal: Large indurated tender swelling filling the ischiorectal fossa. If not drained early, it will extend to the other space forming (Hoarse-shoe abscess). Can be felt only by DRE.
 - 3- Submucous abscess and pelvirectal abscess: tender boggy swelling on DRE.

Complications

(Don't wait for fluctuation☐)

- Otherwise, fistula will be formed. (30-60% of cases)
- Fistula is more common with enterogenic organisms e.g. E-Coli (but extremely rare with skin type organisms)

Treatment:**1) Treatment of the abscess:**

- **Urgent surgery (incision and drainage under general anesthesia).**
 1. If perianal or ischiorectal → drainage under general anesthesia by Cruciate incision with excision of skin edges.
 2. If submucous → drainage through proctoscopy.
(If it is after injection sclerotherapy, it usually resolves spontaneously.)
 3. If pelvi-rectal → transrectal drainage.
- The wound is packed with daily dressings.

2) Treatment for the cause:

- _ (associated fissure) → Lat. Sphincterotomy

3) Treatment for complications:

(Fistula)

- Low fistula → Lay open fistulectomy.
- High fistula → 2 stages operation

Volvulus

Definition

- Twisting of a loop of the bowel around an axis of its own mesentery

Pathology

- Volvulus produces a combination of obstruction of the closed loop type and occlusion of the main vessel at the base of the involved mesentery.
- In a closed loop, the pressure rises rapidly → gangrene & perforation.

A. Volvulus of the Pelvic Colon

Predisposing Factors

- Sigmoid volvulus is common in **elderly chronically constipated males** due to:
 - a. Long sigmoid colon.
 - b. Narrow base of sigmoid mesocolon.
 - c. Heavily loaded sigmoid as a result of chronic constipation.

Pathology

- The loop may rotate $\frac{1}{2}$ turn up to $1\frac{1}{2}$ turn which leads to:
 - Occlusion of veins → congestion.
 - Occlusion of the arteries → gangrene & perforation.

Clinical Picture

Type of patient

- Elderly constipated male with repeated episodes of abdominal pain

Symptoms

1. **Pain**: sudden severe colicky pain.
2. **Marked distension**: in the flanks from Lt. side towards the umbilicus.
3. **Constipation**: absolute but bleeding per rectum may be present.
4. **Vomiting**: delayed for 1-2 days.

Signs

1. **Inspection**:
 - Distension & visible peristalsis.
2. **Palpation**:
 - **Tense balloon of the volvulus** can be felt → going towards umbilicus.
 - Guarding, rebound tenderness if complicated.
3. **Percussion**: Tympanitic abdomen.
4. **Auscultation**: mad abdomen.
5. **DRE**: empty rectum.

Complications and Causes of Death

- Shock & toxemia from gangrenous segment

Investigations

For diagnosis

- **Plain X-Ray of the abdomen**: omega sign.
- **Barium enema**: Ace of spade deformity.

For complications:

- **CBC**: anemia.
- **KFTs**: pre-renal failure.
- **Serum electrolytes**.

Treatment

1. Preoperative preparation "resuscitation"

- NG suction "Ryle".
- IV line
- Catheter.
- Monitoring.

2. Conservative Treatment

- Indications:
 - Early non-complicated cases (i.e. no evidence of gangrene).
- Method:
 - A **rectal tube** is passed through a sigmoidoscope to untwist the sigmoid loop.
 - If successful: **the tube is left in place** and the patient is prepared for later elective surgery.
- Success is confirmed by: gush of gases & fluid stools.

3. Surgery

- Indications:
 - Failure of the conservative treatment.
 - Late complicated cases.
- Method:
 - 1- If the colon is viable and short → Fix to the posterior abdominal wall (colopexy)
 - 2- If the colon is viable & long → Mickuliz or Hartmann's method.
 - 3- If the colon is gangrenous → Mickuliz or Hartmann's method.

4. Postoperative care

- NPO
- Analgesics
- Sedatives.
- IV fluids.

B. Volvulus Neonatorum

- Etiology: incomplete rotation of the midgut that should occur during intra-uterine life.
- C/P: usually baby in the 1st few days of life.
with bile stained vomiting & abd. Distention.
- TTT: Urgent surgery is needed to untwist the gut, divide the band and to broaden the base of its mesentery by further displacement of the cecum to the left.

Mesenteric Vascular Occlusion

Definition

- It is occlusion of the superior (or rarely the inferior) mesenteric vessels or one of their branches.

Pathology

- Arterial obstruction** → ischemia of the mucosa → ulceration and sloughing in 3 hours → peritonitis, after 6 hours gangrene occurs (bacterial translocation).
- Venous obstruction** → gangrene is more delayed than arterial obstruction.
- Reperfusion damage** → the return of blood flow containing bacterial toxins & oxygen free radicals from this segment → to systemic circulation

Causes

- Arterial embolism** (source: MS + AF, MI + Mural thrombus).
- Arterial thrombosis**: due to atherosclerosis
- Venous thrombosis**: due to portal hypertension.

Clinical Picture

Symptoms

- Pain: severe (acute abdomen) → out of proportion to physical signs and not relieved by NG suction.
- Bleeding per rectum when mucosal infarction has occurred.
- Vomiting and diarrhea may be present.
- In late cases patient may present with shock and toxemia.

Signs

If neglected

- General**
Shock and toxemia
- Local**
-Guarding, tenderness and rebound tenderness.

Investigations

➤ For diagnosis:

- Mesenteric angiography or duplex scan**: The value is controversial as it may delay surgical interference.

Treatment

1. Preoperative Preparation

R&M + heparin infusion.

2. Urgent Operation

- Gangrenous intestine**: Resect the gangrenous part + 1ry or 2ry anastomosis.
- Viable intestine**:
 - Embolism** → Embolectomy by Fogarty catheter
 - Thrombosis** → Streptokinase or Bypass.

3. Postoperative care

- The patient is given postoperative heparin and discharged under oral anticoagulants for at least 3 months to prevent recurrence.
- Sedation, NPOI, IV fluids

Paralytic Ileus**Definition**

- It is failure of neuromuscular mechanism leading to failure of peristaltic waves of the intestine (functional I.O.) with patent lumen.

Etiology

1. **Reflex inhibition of intestinal motility** → After major operation (commonest), labour.
2. **Toxic inhibition** → Due to peritonitis, typhoid.
3. **Metabolic abnormalities** → due to hypokalemia, uremia and DKA.
4. **Drugs** → overdose of Anticholinergic or TCA.

Pathology

Failure of transmission of peristalsis → Accumulation of gas & fluid inside intestine → distension → more paralysis & so on.

Clinical Picture

History of the cause e.g. 3rd day Postoperative[□]

Symptoms

1. **Distension.**
2. **Absolute Constipation.**
3. **Vomiting:** repeated effortless.
4. **Pain:** No abdominal pain[□] except for the cause e.g. at site of abdominal incision.

Signs

- **General:** shock + C/P of the cause, e.g. uremia, hypokalemia.
- **Local:**
 1. **Inspection:** distension, scar of previous operation
 2. **Percussion:** Tympanitic abdomen + Pseudo-shifting dullness.
 3. **Auscultation:** dead silent abdomen

Investigations

See before

Treatment**A. Prophylaxis (avoid the predisposing factors)**

1. **Preoperative** → correction of the electrolyte imbalance.
2. **Intraoperative** → gentle manipulations.
3. **Postoperative** → NPO till intestinal peristalsis is regained.

B. Active Treatment**A- Resuscitation and monitoring:**

1. **Ryle's** tube → GIT suction to relief the distension.
2. **IV line:** Restoration of fluid & electrolyte balance+Antibiotics.
3. **Sedation** → Pethidine 100 mg IM.

B-Treatment of the cause:

e.g. Drainage of peritonitis, TTT of internal hemorrhage.

C- Observation for the signs of recovery:

- which are:
 - Pain
 - Passage of flatus
 - Distension

D- Post-operative care: Sedation +NPO+IV fluid

Intestinal Obstruction

Intussusception

1. Primary Intussusception

Definition

- Invagination of an intestinal segment (intussusceptum[□]) into the lumen of an adjacent one (intussusciens[□]) or telescoping, due to unknown etiology.

Pathology

- An intussusception is composed of:

1- Entering or inner layer.	}	intussusceptum
2- Returning layer		
3- Outer sheath	}	intussusciens
- The blood supply of the inner layer is liable to be impaired at the neck of intussusception.
- Ileocecal:** (75% → commonest[□])

Etiology

Infantile intussusception (idiopathic)

- Possible cause is adenovirus
- Evidence:** frequent occurrence of intussusception at age of weaning[□], summer (due to increased incidence of gastroenteritis).

Clinical Picture of Infantile Ileocaecal Intussusception

Any infant having colicky abdominal pain with passage of blood-stained mucus per rectum should be suspected of having intussusception until proved otherwise

Type of Patient

- Usually a healthy male between 3 – 12 months old(male: female= 2:1).
- With history of weaning or recent gastroenteritis (caused by adenovirus).

Symptoms

- **Pain:** in attacks with crying, screaming and drawing up of legs.
- **Vomiting:** projectile vomiting occurs in 85% of cases.
- **Absolute constipation:** The child passes blood & mucus per rectum which is called (Red Currant Jelly stools[□]).

Signs

- **General:** dehydration(sunken eyes, depressed fontanelles, dry tongue & inelastic skin)
- **Local:**
 - **Inspection:**
 1. Visible peristalsis may be observed.
 2. Distension of the abdomen (usually absent in early cases).
 - **Palpation:**
 1. Sausage-shaped mass
 2. Signe de Dance → right iliac fossa feels empty.
 - **Percussion:** Tympanitic abdomen.

- **Auscultation:** mad abdomen.
- **DRE:**
 1. The finger is stained with mucous & blood.
 2. Apex of the intussusception may be felt.
 3. Very rarely, intussusception may protrude through the anus.

Complications and Cause of Death

- Shock & toxemia from gangrenous segment (1ry intussusception is prone to early gangrene than 2ry intussusception).

Differential Diagnosis

1. **Acute enterocolitis:** Faecal matter is present together with the blood & mucus.
2. **Rectal Prolapse:** A finger can be introduced into the rectum along the intussusception, but in prolapse the finger can not be introduced
3. **Henoch's purpura:** Purpuric rash is present; it may be associated with intussusception which will need operation

Investigations

➤ For diagnosis:

- **Plain X-Ray:** see before
- **U/S with saline** → Target sign
- **Barium enema (Diagnostic & Therapeutic):**
 - It shows the "Claw sign"☐.

Treatment

Urgent operation after preparation

A. Preoperative Preparation

- IV fluids and antibiotics, NG suction, antibiotics and monitoring.

B. Hydrostatic Reduction☐

- **Indications:** only early non complicated case using barium enema.
- **Success of reduction is confirmed by:** Filling of the terminal ileum, caecum & appendix (no claw sign).

C. Surgical Reduction

- **Indications:**
 - 1- Late complicated cases from the start.
 - 2- Failure of hydrostatic reduction.

If the intussusceptum is irreducible or gangrenous, resection & end to end anastomosis

- **Recurrence:** avoid by stitching the terminal ileum to the caecum.

2. Secondary Intussusception

Types

- 1- **Ileocecal:** (75% → *commonest*☐)
- 2- **Ileoileal.**
- 3- **Ileocolic:** more liable for strangulation.
- 4- **Colocolic.**
- 5- **Retrograde.**
- 6- **Multiple.**

Etiology

1. Polyp.
2. Submucous lipoma.
3. Papilliferous carcinoma.
4. Inverted Meckel's diverticulum.
5. Submucous hematoma in Henoch Schonlein's purpura.